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EDITORIAL

INTRODUCTION

THIS special edition of The American Journal of Surgery is designed to appeal more to the practical surgeon than to the surgical scientist. It is neither the hope nor intention of the Guest Editor to develop anything new or unusual in the field of traumatic or industrial surgery.

Across the desk of the Guest Editor for many years has flowed an array of traumatic problems; problems involving not merely the purely scientific aspects of traumatic surgery, but the administrative, medicolegal and philosophic as well. He has seen physicians in every professional stratum struggle in the quest for good end results in types of patients who in many instances, because of class consciousness, avarice, or vindictiveness, presented grave psychological handicaps to full recovery. He has seen these struggles terminate, in very many instances, in a triumphant manner, a tribute to the skill and understanding of our profession.

In many instances, however, whether because of the inherent nature of the injury, the limitations of the surgeon, or the short-comings of the patient, the end results have been less satisfactory. The Guest Editor has therefore drawn freely from his own experience in an effort to assemble here a symposium dwelling upon those injuries and their accompanying social and economic aspects that may have contributed to such unhappy end results.

With the development of mechanized industry, the growth of motor transportation, and the increasing social awareness of our people, the surgery of trauma has come to occupy an increasingly conspicuous place in our professional activities. Many, if not most, traumatic injuries assume a legal as well as a medical significance in these days of workmen's compensation and growing court dockets. The surgeon is now being placed more often in the position of having to exhibit, and often defend, the results of his handiwork before

judicial and quasi-judicial bodies. He is brought into intimate contact with industry. He must learn either to adapt himself to the needs and requirements of industry, or else relinquish participation in a most interesting and lucrative field of practice. This involves, among other things, the maintenance of practical relations with insurance companies, without whose participation the execution of the principle involved in workmen's compensation would be impossible, and without which the just placing of responsibility for highway accidents could often not be accomplished.

In this issue of The American Journal of Surgery, therefore, an effort has been made to clarify relationships involved through workmen's compensation and medicolegal problems in general.

It is, of course, impossible to embrace in one issue, even as large an issue as this, a full coverage of all of the intricacies involved in the surgery of trauma. The surgeon is confronted not only with the usual professional problems, but many others of special nature, which in other types of medical practice are of slight significance.

It is hoped that this issue will contribute to the need which exists in medical circles for knowledge of the subjects represented.

ALLEN D. LAZENBY, M.D.

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ORIGINAL ARTICLES

THE FUNDAMENTAL PHILOSOPHY OF WORKMEN'S COMPENSATION

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NEW YORK CITY

MANY of those now actively concerned with workmen's compensation laws, either administratively or in the medical and surgical field related to them, or otherwise, have been so concerned for less than the brief quarter century that has seen the adoption and growth of such laws, and, consequently, are little acquainted with industrial accident systems that preceded this compensation era. Moreover, many of us who have been in contact with such laws since the beginning are prone to consider only the advantages and the weaknesses as they now exist, and too frequently are unmindful of the system that preceded compensation, or the reasons that underlay the demand for a compensation law.

For a correct understanding, therefore, of the fundamental philosophy of compensation laws, it is needful that we review briefly the history and philosophy of the laws relating to the employer's liability for injury to his employees.

At the outset we may accept the fact, so often mistaken today, that compensation was not in its original concept regarded as "social insurance" nor as part of a larger program of "social security," although the acceptable working of the system may have had much to do with creating the demand for the latter. Compensation was, on the other hand, established as a substitute for the then existing "employers' liability" laws. The system of laws commonly denominated as "employers' liability" was not itself an old system. It had developed from a much broader view of the employer's liability to his injured employee than had theretofore existed. Those laws were actually looked upon as "liberal" at one time.

For what did they supplant? If we go back far enough, there was no liability on the part of the feudal lord for the injury of his retainer, whether serf or freeman, and his care of such retainers was purely within his grace and discretion. Organized facilities for such care were unknown and nonexistent, and, in case of injury or disability from any cause, one was largely dependent upon the voluntary assistance of neighbors and clansmen. And this conception and practice persisted for centuries. Not until comparatively recent times did a different conception arise. After the birth of the industrial era, in the mid-eighteenth century in England and later in France and Germany, with a more concrete organization and with the then modern ideas and methods, men first began to assert liability of their employers for damages in case of injury and to demand their rights in the courts.

Our common law was derived from the English law, and the only rules the courts in England then knew as to liability were the principles of the old Roman law, which applied as between strangers, not as between an employer and his employees. Under these principles the employer was held liable only for the results of his own personal wrongdoing. He was not liable for injury of his employee due to the fault or

neglect of a fellow workman, but each employee was deemed to have accepted the risk of the employment when he voluntarily accepted such employment. Negligence on his own part, if contributory to the injury, was a bar to any recovery against the employer.

The rigorous application of these principles soon came to be recognized as socially deplorable and to work hardship and injustice in many cases.

Consequently there succeeded a disposition on the part of legislatures, and eventually of the courts, following different courses in different jurisdictions, both in this country and abroad, to mould this law and the application of its principles, in efforts to make it fairer to the workmen. The narrow rules of proof were variously modified, the employer's defenses were cut down, the faults of employees came to be imputed to the employer when they resulted in injury to another employee.

New doctrines came to be voiced both by courts and by statute; growing difficulties ensued, delay and expense were common and created hardship for both. Fanciful and ofttimes wholly impractical obligations were laid on employers for the express and avowed purpose of erecting new rules of liability. Notwithstanding these arbitrary rules, injured workers, or their legal representatives when the workers had been killed, found themselves excluded from any remedy in many cases where justly they should not have been excluded. On the other hand, in many cases, employers felt the harshness of recoveries based upon such arbitrary rules. A growing impatience with the system began to manifest itself in this country not long prior to 1910. Previously in England and on the continent of Europe a new system had begun to function and to make itself felt.

Legislative committees and research investigators had begun to expose the glaring faults and inequities of the employers' liability system. Chief among the evils, in addition to those just cited, was the fact that in all disputed cases attorneys at law were essential in the assertion of rights in the courts; the workers were generally unable to pay retaining fees, and hence the contingent fee came into vogue. Under agreements for such fees, the attorney became, in effect, the partner of the injured person; he had an absolute right to share in any recovery, generally taking one-half of the recovery for himself and often an additional sum for his expenses. The injured person, perhaps after years of litigation, got what was left, and too often was prone to wonder who it was that sustained the injury.

And what of the doctor and what of medical care and treatment? Singularly enough, in all the centuries of development of the principles of employers' liability, no provision ever had been made for medical care, other than that in some situations the employer was expected at his own expense to provide first aid.

The injured person was expected to provide himself with medical care. The doctor and the hospital had to content themselves with the promises of the patient to take care of their fees out of any recovery, and the cost of such treatment was a proper item of damages to be proved in the trial. If the case actually came to trial, the doctor fairly could protect himself, for his testimony frequently was an essential part of the proofs. When, however, a private settlement was arranged, which was the rule in a large number of cases, the medical bills were frequently forgotten. Moreover, in a very large percentage of cases, no negligence of the employer could be shown, and hence no recovery could be had. Then the doctor's part was pure charity. Under such circumstances it was not to be expected that treatments would be of the best or that the more skilled and experienced surgeons would be attracted to the work.

In these days when so much is heard of the hardships imposed upon the medical profession in compensation cases, it is interesting to note the vast improvement in

the matter of industrial medical and surgical care that has developed in a space of time embraced within our own active association with the problem. From a system where the lawyer got much and the doctor got little, we have evolved a system where the lawyer complains of his negligible returns, while the doctors now receive for medical and surgical care many millions every year.

The first workmen's compensation law to be enacted in this country was in 1911, and the system rapidly extended to many states, until now it is the law in all but two states, in the District of Columbia, in our island possessions, and in the Federal field of longshoremen and harbor workers.

In no two jurisdictions is the law precisely the same, and there are very wide variations between some. In view of the wide differences, we may fairly ask what it is that distinguishes the compensation system in the different states and makes it common in many of its attributes.

In the first place, it is a workmen's system and is a "beneficial" law for the workmen. The relation of employer and employee must exist before the compensation system comes into play. The fact of "employment" is jurisdictional and basic. Often there is heard complaint in specific cases where compensation for disability is denied or charges for services, medical or otherwise, are disallowed. If the relationship of employer and employee did not at the time of accident legally exist, no other decision could be made. Sometimes the person injured was in fact a trespasser on the employer's premises, or perhaps a mere licensee permitted on the premises for some purpose of his own, as for example, when he entered the premises looking for work. Then again, as often happens, he might be an independent contractor within the meaning of the law and not an employee at all, although having many of the characteristics of an employee.

Having established in a particular case that employment exists, it is necessary that the facts show the injury to have "arisen out of and in the course of" such employment. This rule of responsibility is common to most compensation laws, though some provide that the injury need only arise "in the course of" the employment. In one sweeping gesture, by this clause, industry is made to assume the responsibility for virtually anything that may happen to the worker in the course of his work. Of course, with such an all-inclusive rule of responsibility it was necessary to enact in the statute certain exceptions. These exceptions, generally expressed, are that no compensation is payable if the injury was occasioned by the worker's own wilful misconduct or his intent to injure himself or another, or if the injury was caused solely by his own intoxication. The necessity for these exceptions, of course, is apparent.

What constitutes an injury that "arises out of and in the course of" employment is one of the most controversial questions that is presented by the compensation system. So broad a rule is it, one might assume that it practically would remove all controversy. It is probable, however, that more court decisions are involved in the interpretation of this expression than in any other that presents itself. For instance, what does "in the course of" employment mean? When, for the purposes of the compensation law, does employment begin and end? Does it begin when a man leaves his home to go to work, or does it begin only when he is at his actual work place? Suppose he is assaulted by strikers as he leaves his home and before he arrives at the job? And what of the salesman who leaves his home to journey by train, by air or by motor, and whose day is filled with all sorts of incidents, including the incidents of living in hotels, attending perhaps sports by day and theaters or pictures by night? While thus engaged, is he in the course of his employment and entitled to compensation? Where is the line to be drawn? When, too, does a worker take himself out of his employment by some

act of his own, even though in the usual hours of his work? These are but a few suggestive questions that present themselves and have been the subject of litigation.

An even wider range of questions is suggested by the phrase "arising out of" the employment. Does sunstroke, occurring in the employment, arise out of it? Or is that an elemental thing to which all are subject irrespective of employment? I may remark that the court holdings on this question are not all alike. In exactly the same category is the question of frostbite. How far may a worker go to satisfy a natural curiosity as to a matter not of his particular concern and still have a resulting injury give rise to compensation? These are but suggestive of innumerable questions that arise. The fact that an injury arises out of some happening that is "incidental" to the employment allows of great controversy as to whether the act is or is not fairly incidental.

Involved also is the troublesome definition of what does the term "injury" mean.

Broadly, injury is limited to "accidental injuries" or "injuries by accident." Both expressions are used, depending upon the law of the particular state. Nor are the two expressions always held to mean the same thing.

Universally, however, the term injury is held to include such disease or infection as naturally or unavoidably follows as a sequence of the injury. This, of course, is only common sense, but in carrying it into effect, we reach some rather startling results. Out of this principle of the law has arisen the very elastic and originally unexpected theory of the "aggravation, acceleration, exacerbation or activation" of disease. Surely the original authors and adaptors of the compensation system did not visualize the extent to which the use of the term has made industry responsible for disability and death from conditions of disease that antedated some accidental occurrence and was perhaps only speculatively affected by it. We have seen cancer, both carcinoma and sarcoma, aggravated by trauma—ever so slight; Hodgkin's disease accelerated; tuberculosis activated—all to the point where now it becomes the responsibility of medicine, by research and compilation of case data, and by round table discussion, to establish the criteria by which it may fairly be adjudged whether morbid conditions of the body have become disabling factors by reason of industrial trauma. To what extent does "lowered resistance" following an accident contribute to the contraction or activation of disease?

Without exception, and this is one of the most distinguishing features of the compensation system, the fault or negligence of the employer or the fault or negligence of the worker, does not enter into the question of liability for compensation. The entire absence of fault on the part of the particular employer will not defeat the right to compensation if the worker met with an accidental injury that arose out of and in the course of his employment. Rather would it seem that fundamentally the industry as a whole, rather than the particular employer, is held to be at fault, if philosophically fault need be implied.

Certainly, viewed from the standpoint of its economic effects, it matters not at all if the accident causing the injury was due to the employer's negligence, the worker's own carelessness or the fault of a fellow employee. Nor does it matter if the accident was preventable or inevitable. This latter phase is, however, of signal interest to the industrial engineer and safety man.

We might, of course, ask what is an "accident." That question has been asked of the courts in many cases upon many and varied states of fact. Is it only the unforeseen, the fortuitous happening, or may it be the expected result of an intended act—as for example, an assault? And must it happen suddenly? Would exposure to a deleterious gas from a leak, for many hours or days, be an accident?

Another distinguishing feature of compensation is that it does not provide damages, on a speculative basis, but is limited to a fixed percentage of the loss of earnings or earning power. Nor is this ordinarily paid in one sum and the matter completed, as under the old liability system, but it is made payable during the continuance of the disability, usually with appropriate maxima. Provision is also made for graduating the compensation rate downward when a partial earning capacity is established. And except with respect to death benefits and those cases where there has been an amputation or permanent partial disability, compensation ceases when a full earning capacity is restored.

Of prime interest to the medical profession is the question thus suggested, namely, what constitutes disability and how shall it be determined? The procedures for such determinations are varied. If fairness and the appearance of fairness are to be preserved, medical examinations should be so far as possible made by impartial examiners. Certainly as to disputed cases this should be the rule. Great difficulty is encountered in those cases where the complaints are wholly or largely subjective. And the difficulties are immeasurably increased where the complaints are neurologic or hysterical or susceptible of exaggeration. Traumatic neurosis as a pathologic entity was not unknown to us before compensation days but we have surely become quite familiar with the condition in recent years.

Possibly the greatest single principle underlying compensation, from the standpoint of the worker, is not so much the money benefits paid during disability, vitally important as that is. Rather the greatest benefit is the obligation placed upon the employer to furnish necessary medical and surgical treatment in the event of an injury. More than in any other way this requirement marked a great forward step over the old employers' liability system. How logical it is, and yet how difficult it became to bring the realization of this principle home to employers and to legislators! Here again there is not entire uniformity among the states. In some few states the obligation for medical care is unlimited as to time and amount, the only limitation being "reasonableness." In some states the period during which treatment shall be furnished by the employer is prescribed by law, just as though by law a fixed time limit could be set within which a wound with all its complications must heal. Sometimes the maximum cost of medical treatment is set at a predetermined sum; while in yet other states a double maximum is set, one as to length of treatment and the other as to amount. Many states have provisions under which the time limit can be extended by the administrative authority when the need for extension is shown.

Hospitalization, of course, and necessary nursing are also required by law.

The matter of the selection of the physician or surgeon to treat a case has ever been a difficult one.

The first compensation laws, with hardly a variation, required the injured employee, if he would avoid payment of his medical cost himself, to accept the services of a physician designated or authorized by his employer. The theory of this was logical. It proceeded on three major assumptions: first, that the employer, generally speaking, was better informed and hence better able to select a competent doctor for the injured person; second, that being required to pay the bill, the employer should have something to say about the person to be selected, that his cost should not be uncontrolled; and, third, that, to state it bluntly, the employer's interest is to restore the worker to full earning power as soon as possible, since he is charged to make compensation during disability. His self-interest in the compensation payable, should dictate the selection of none but competent physicians, and, therefore, the injured man benefits. The right of the employer to select the physician was contemplated as an obligation, which, if

wisely exercised, would have resulted to the benefit of the worker, as well as in the interest of the industry.

There is a growing sentiment in some sections of the country that this system has outgrown its usefulness, and one of the most highly controversial questions today is the question of giving to the injured man greater latitude in the selection of a doctor to treat him. It is not the purpose of this paper to enter into that controversy. Much is being said on both sides of the question by those who have a direct interest or relation to the problem. As so often happens in such controversies, exaggeration and overstatement are not foreign to the discussions. In the light of the history of medical treatment in compensation, the growth of it in volume and amount, and the certain payment of bills in valid cases, it seems legitimate to make comparison of the economic contribution of the doctor to the problem today as compared with his relation to the old employers' liability system.

Another fundamental should be mentioned, that is, the employer's responsibility for the disease of his employees. Under our old employers' liability system it is very questionable if the employer had any responsibility as to health. There seems to be no case in the English courts wherein is recognized any such liability at common law. With the development of modern industry, with the use of various toxic and poisonous substances, and with the general recognition of the employer's responsibility for accidents, there has come about a growing recognition of the liability of the employer for disease. Such diseases are commonly known as "occupational diseases." The term is one that almost defies definition. And yet define it we must, either in words or by the accepted principles laid down by the courts in specific cases. About one-third of the states make some provision for occupational diseases. These provisions differ widely.

Obviously, all diseases are not occupational diseases. The definitions in the laws of Michigan and Rhode Island come most nearly to expressing what we mean by the term. Those two states define the term "occupational disease" to mean only a disease that is due to causes and conditions "characteristic of and peculiar to" the trade or employment. In other words, a disease to be "occupational" must be the result of a "trade risk." This does not mean *any* disease of ordinary life speculatively related to an incident of the employment.

In the extension of laws for occupational diseases to other states, we should always keep before us this distinction. To include all ordinary diseases just because they happen to workmen, is to provide general health and life insurance at the expense of industry.

Perhaps there is no more misunderstood factor in the compensation system than the relation to it of insurance. Insurance did not seek out compensation, nor seek to absorb the compensation system as a means of making a profit. Insurance has, however, immeasurably contributed to and become an essential part of the system. It was early recognized that "security" for the payment of compensation was a prime essential in the system if it was to be workable. With compensation payments extending for years into the future, as, for example, the pension payments to a widow or to a worker permanently and totally disabled, some security other than the questionable responsibility of the employer, who might next year go out of business, must be devised. Insurance formed the only basis for such security within reasonable limits. Funds must be set aside at the occurrence of an accident to provide for the payments which may be required over a period of years. Only by a system of insurance, with the cost to industry based upon the actuarially predetermined estimates of such cost, as reflected by the combined experience of all industry, and with strict supervision of reserve funds, can the workman be assured that he exchanged his former speculative but unlimited right

of recovery for a better system that will afford him security and certainty in the event of injury, even though his right of recovery be limited by law. The results of this new system are seen in the enormously increased payments of benefits to injured workers and the dependents of those industrially fatally injured.

Another distinguishing characteristic of the compensation system is its general freedom from legalistic and technical procedures for the adjudication of rights and liabilities under it. The evils that attended the former system, with its delays, its contingent fees for lawyers, the substitution in many cases of compromise settlements, often woefully inadequate, and the reliance upon technical rules of evidence to defeat otherwise meritorious claims, reacted powerfully to bring about a new era in the field of compensation for industrial injuries. While resort to the courts for the enforcement of compensation claims is still the rule in a very few states, the more common and usual procedure is to entrust to industrial accident boards and commissions the administration and enforcement of compensation laws. These boards, acting in a quasi-judicial capacity, are usually not bound to apply technical rules of evidence or procedure. In many states their fact-finding power is final. Through their operations attorneys are not necessary in any but the exceptional case. The effort seems to have been successful to establish a method of adjudication that is informal, non-technical, summary and speedy. The workman gets quickly what is his due and gets it all. He gets it when he most needs it, that is, while he is incapacitated. The trend of the moment toward injecting lawyers into compensation procedures, with the almost inevitable result of introducing technical and legal elements, is one to view without great enthusiasm, if not with actual distrust.

Of the greater benefits of the compensation system over the old employers' liability system there can be no doubt. Imperfect, no doubt, but that is a human failing. But the general recognition of the fundamental principles underlying the compensation system has made possible the growth and development of modern industry. Ofttimes the voice of the experimentalist is heard suggesting how this system can and should be changed by extensions in many new and untried ventures. When such experiments are proposed, let us always apply the fundamental principles as we have understood them and as they have developed our present system. By that rule we shall be enabled to judge correctly the value of those proposals. But let us never lightly exchange what we have developed so usefully for the dreams of an Utopia that have no basis in sound and fundamental principles.

MEDICAL REPORTS BORE ME, TOO

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PRESUMABLY the word "bore" doesn't begin to express the feelings of the physician who is required to make out a medical report and anyone else who in the course of his duties must read it. With humble apologies to the "Stroud twins" we might go on to say that medical reports not only bore all who have anything to do with them but they displease, annoy, trouble, perplex, rag, tire, irk and so on, ad infinitum.

Not long since, I learned that at a meeting of a hospital board a physician was called upon to report on a case which he had in the hospital. He was a well-trained physician of long experience, chairman of the Board of Health in the particular locality in which the hospital was located. He opened his remarks relative to the case with the following words: "Gentlemen, this is not a very good record of the case. I hate to write medical reports just as much as you all do."

Is not this statement indicative of the reason perhaps why the writer has been asked to prepare this article? But, why should this be so? While it has been agreed that medicine is not an exact science, medical men do represent a highly trained and reasonably scientific profession. Perchance the answer to this question may, after all, be relatively simple, e.g., that no one has previously taken the time and trouble to explain what "all the shooting" is about when the business of insurance complains that medical reports are a bore. No one has explained to the medical profession the use to which these reports are put or explained how valuable from the standpoint of the patient, the administering accident boards and commissions, and insurance carriers, these reports actually are.

Some physicians may feel that the insurance companies have little right to require as a precedent to the payment of physicians' bills an adequate medical report, or in fact question if the insurance carrier has any right to a report at all. In business, and presumably in the professions, all parties strive to please valued customers or clients.

According to Best's Insurance Reports for 1937, we may well assume that between 45 and 50 million dollars annually are being paid out by the stock and mutual insurance companies to physicians and hospitals for professional services rendered in compensation cases.

In addition to this vast sum, millions more are presumably paid out by the employers who do not insure their compensation liability, and if we were to venture outside of the field of compensation insurance and consider the amount paid by casualty companies in other forms of coverage and by the life companies in their life extension work, etc., etc., we think that possibly we have made out a case proving that the business of insurance is a worthwhile client whose request for coöperation should not fall upon deaf ears or sightless eyes.

The aim of this article is not to be controversial or argumentative, but helpful. After nearly forty years' experience the author still feels that the difficulty in securing prompt, adequate and proper medical reports is due to lack of understanding, on the part of the medical profession, of the problem.

We may break this subject down into several parts. First, why are satisfactory medical reports difficult to obtain? Let us revert to the yesterdays of a quarter of a century ago when compensation laws were

first being considered by the legislatures of some of the states; to that time when this great piece of social legislation appeared to be so very simple of administration, so devoid of procedural difficulties, when it seemed that following an injury arising out of and in the course of employment all that would be necessary for the employer or his insurance carrier to do was to pay the amount of compensation to which the injured man was entitled, as decreed by the law and defined as respects period of time and amount. Because this law which placed upon industry financial obligations without regard to the question of fault, a thing altogether unheard of up to that time in this country, it established an entirely new relationship and additional responsibility as between the employer and the employee, the doctor and his patient. The safety movement already in existence received enthusiastic impetus, but always lagging behind in the progress of this social legislation has been the development of a proper medical program of coöperation.

It would serve no useful purpose at this time to hold an inquest to determine who was to blame or to attempt to narrate seriatim the progress which has been made to date.

Exception to this might be made to pause briefly in our discourse to pay tribute to those whose breadth of vision conceived and brought into being a new branch of surgery, or specialty if you will, generally referred to as industrial surgery.

Though it seemed heresy to some of the medical profession even to consider that surgery as applicable to industrial injuries could possibly be any different from the surgery which always had been, much credit is due the forward-looking members of the American College of Surgeons for their championing of the cause of this phase of surgery and especially for their progressive program as respects the treatment of fractures which has always been a serious and expensive problem to carriers of compensation insurance.

We have referred to the change in the relations between the physician and his patient. Under the Workmen's Compensation Law it was necessary that the physician must reveal to some third person, e.g., the employer or the insurance carrier, the facts relating to the injuries suffered by his patient, i.e., their seriousness, the prognosis as respects disability and his estimate of any permanent impairment that might reasonably result. So, it may well be that the failure to appreciate or unwillingness to conform to this changed relationship may be to some considerable extent responsible for the difficulties which we experience in securing prompt, adequate and proper medical reports in compensation cases.

Why are medical reports necessary? Who uses them and how? They are necessary because in accordance with the provisions of all compensation laws no compensation is due the injured man, generally speaking, unless he has suffered a disabling injury requiring his absence from work or a permanent impairment which might possibly result from an injury in which there was no disability (although such a situation in these days would be extremely rare). It is necessary that these reports be rendered promptly due to the fact that compensation is in part presumed to take the place of the pay envelope whose arrival usually ceases with the beginning of absence from work.

Who uses them? First of all, the employer or the insurance carrier must have them for the following reasons:

First, to determine if the injured workman will be disabled a sufficient length of time to entitle him to the benefits provided by the Workmen's Compensation Law.

Second, to determine the probable length of that disability.

Third, to determine if their will be any permanent impairment, e.g., amputations, stiff joints, etc.

Fourth, that the carrier may at the earliest possible moment erect a suit-

able reserve—later on we will refer to what this reserve is.

Now the second group that are interested in the prompt receipt of an accident report are the industrial accident boards or commissions whose responsibility it is to administer the laws of their respective states. Some of these boards or commissions require that medical reports be filed with them. Incidentally, some require that medical bills be referred to them for approval before payment. It is the function of these boards or commissions to satisfy themselves that the injured employee has been justly dealt with, that he has received the compensation which is provided for by the compensation law. These groups oftentimes arrange that impartial examinations be made of the injured in order to satisfy themselves impartially that the medical facts are as have been presented by the carrier or the employer.

Who else is interested in these reports? In serious cases probably the most interested individual is the medical or surgical director of the carrier who must, through the picture painted in the report, determine if there is anything that the carrier can do through its medical department to assist the attending physician—and, we are happy to say that through the years there has been a growing inclination or willingness on the part of the medical fraternity to consult with and ask for suggestions from the medical departments of the larger companies writing compensation insurance, an indication of coöperation which is indeed cheering and promises much for the future.

Perhaps the next phase we should consider is the form to be used by the physician in making out the medical report. A few boards or commissions have a stock form of report which they require shall be used. In most cases that form was merely for the convenience of all concerned, but as time went on and our interest in medical matters improved or grew, the inadequacy of those reports was recognized not only by the carriers, but by the International Association of Industrial Accident Boards and Commissions. Each group appointed a Committee on Forms part of whose duty was to attempt to standardize the many forms required by each state in the administration of this law. With a fine spirit of coöperation these Committees met separately and later in joint conference and among other forms attempted to standardize on a medical report form. The standard form for surgeons' reports known as "Standard Form 2" was approved by the joint committee and later adopted by the International Association of Industrial Accident Boards and Commissions at their convention in Columbus, Ohio in 1932. This conforms more or less in its details to the requirements of most of the states; it has been officially adopted by many states and others, while they have not adopted the form, have not objected to its use. We shall attempt later on to explain the reasons how and why this report should be completed, but before doing so, and to sustain further our contention as respects the need for adequate medical reports, we should like to give some illustrations that have come to our attention from time to time which have so clearly indicated the need for complete, adequate and accurate medical reports. While the cases are from our own records, for obvious reasons they will not be identified in this paper.

Case I. In this case the Physician's Report (Form SF2) stated in answering question 6: "Crushing of second finger on left hand. End of finger back to the first joint entirely amputated." Upon that information, compensation in accordance with the provisions of the law was paid to the injured man. Subsequently, we learned from information obtained from the doctor who made out the first report that only the tip of the bone of the terminal phalanx was actually lost and therefore there was no compensation due.

Case II. This was a colored boy who had sustained a broken ankle. He was taken to the hospital where he was cared for by his physician. After the patient had been in the hospital about a month he was discharged and a couple of weeks later the physician sent in a report

that the patient was all well and was able to return to his usual work. The injured boy was advised to this effect and he tried to go to work, in fact he did work two or three days, although suffering considerable pain. Several weeks later the young man reported to his employer that he was unable to work because the ankle was still bothering him. Later on *x*-rays revealed the fact that the ankle was entirely unreduced, that it was not united, and that at the time the case was closed by his attending physician the patient was in no way able to return to his work. The result was that the patient had to be turned over to another doctor, the fracture rebroken and many months of disability ensued. This meant increased cost to the employer, insurance company, pain and suffering to the injured man. This well illustrates the difficulties caused by a report that was too optimistic.

Case III. This was a patient with a broken hip. The physician took *x*-rays and found that everything was all right, but three or four months afterwards, without checking his results by *x*-rays, he allowed the patient to get out of bed. The patient walked about, suffering a moderate amount of pain. There were optimistic reports from the physician. At the end of a year the patient had not yet returned to work, but still the reports were optimistic on his condition. Examination by a second physician revealed that there was no union and that the patient had been all of the time walking about on an ununited fracture of the neck of the femur. Result—extensive, long treatment had to be done, which took a great deal of time and caused a great deal of expense.

Case IV. This patient had his arm injured in some gears, sustaining fracture of the hand, wrist, elbow and shoulder. He was under the care of a busy, active surgeon. Repeated efforts were made to secure a medical report from him. We were put off for a period of three or four months. The doctor was taken sick and died before the initial medical report came through on a very sick patient. This was four months after the time of injury.

Case V. This patient had a septic hand. After several weeks of treatment, the hand was getting worse although the medical reports were very optimistic. It was brought out after a long time and many requests for reports that the patient was suffering from diabetes and that this was the reason that the disability was so prolonged.

If these cases indicate the failure of complete and prompt and accurate medical reports, then their citing has been well worth while.

As doctors you have heard a great deal about the insurance company reserves, and it may be of interest to you to learn that one of the tests of the financial stability of any casualty insurance company is the adequacy of its reserves, and that the state insurance departments under the law and in the interest of the public good are constantly on the alert to determine that the companies doing business are solvent.

Generally speaking, in every other business a reserve is an asset but in the business of insurance it is a liability. Because it takes the place of a potential debt, so-called, the amount set aside to liquidate this debt should at all times be adequate.

In order to accomplish this, each claim when it comes into the insurance company offices is given a money value called a reserve.

If you were to build a house and your architect told you it would cost ten thousand dollars, you would see to it that you had that sum of money in the bank to pay for it. The financing of a compensation case is no different from that of financing a house and instead of relying upon the plans of architects we must rely on doctors and surgeons to furnish specifications in the form of medical reports in order that the potential cost of that case can be properly determined. Each case is a separate bookkeeping account against which amounts are drawn for the purpose of making compensation, medical and expense payments. In order that this account may at all times be solvent, we rely to a large extent upon your full coöperation as respects furnishing us with the information requested in the medical reports.

How to Make Out the Report

We hope that in this preamble we have aroused sufficient interest to justify the

Form No. S. F. 2

STANDARD FORM FOR SURGEON'S REPORT

Approved by I. A. I. A. B. C.

State's Number For: | File: Carrier: Employer:

Carrier's File No.

(The spaces above not to be filled in by Employer)

The Patient

1. **Name of Injured Person:** ..... Age: ..... Sex: .....
2. Address: No. and St. ..... City or Town ..... State .....
3. **Name and Address of Employer:** .....

The Accident

4. **Date of accident:** ..... Hour ..... M. Date disability began .....
5. State in patient's own words where and how accident occurred: .....

The Injury

6. **Give accurate description of nature and extent of injury and state your objective findings:** .....
7. Will the injury result in (a) Permanent defect? ..... If so, what? .....
   (b) Facial or head disfigurement? .....
   (Permanent disability such as loss of whole or parts of fingers, facial or head disfigurement, etc., must be accurately marked on chart on reverse side of this report.)
8. Is accident above referred to the only cause of patient's condition? ..... If not, state contributing causes: .....
9. Is patient suffering from any disease of the heart, lungs, brain, kidneys, blood, vascular system or any other disabling condition not due to this accident? ..... Give particulars: .....
10. Has patient any physical impairment due to previous accident or disease? ..... Give particulars: .....
11. Has normal recovery been delayed for any reason? ..... Give particulars: .....

Treatment

12. Date of your first treatment: ..... Who engaged your services? .....
13. Describe treatment given by you: .....
14. Were X-Rays taken? ..... By whom? ..... (Name and Address) ..... When? .....
15. X-Ray diagnosis: .....
16. Was patient treated by anyone else? ..... By whom? ..... (Name and Address) ..... When? .....
17. Was patient hospitalized? ..... Name and address of hospital: .....
18. Date of admission to hospital: ..... Date of discharge: .....
19. Is further treatment needed? ..... For how long? .....

Disability

20. **Patient was / will be able to resume regular work on:** .....
21. **Patient was / will be able to resume light work on:** .....
22. **If death ensued give date:** .....

REMARKS: (Give any information of value not included above) .....

I am a duly licensed physician in the State of .....

I was graduated from ..... Medical School in ..... Year .....

Date of this report: ..... (Signed) .....

This report must be signed personally by physician. Address: ..... Telephone .....

FIG. 1.

hope that the following attempt to analyze or explain in detail the reasons for the questions asked on this report will be careability must be judged in many cases by the type of report you make to the carrier or employer.

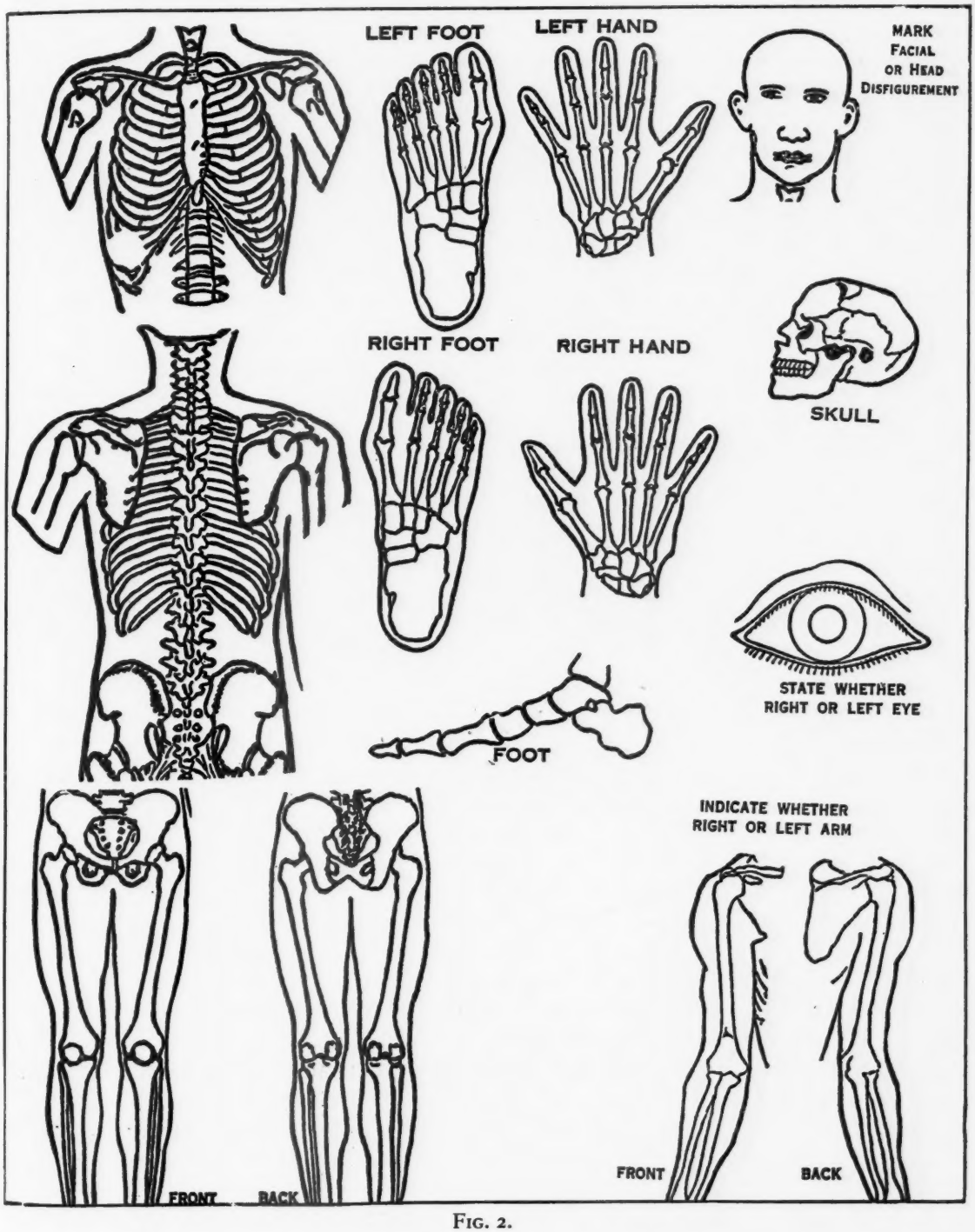

Fig. 2.

fully read and thoroughly digested. We feel sure that if they are, medical reports will no longer bore the maker or the reader. Remember, doctor, that your work and

For your convenience, we have inserted facsimiles of the front and reverse sides of the Standard Form of the Surgeon's Report known as Standard Form 2. (Figs. 1 and 2.)

FIRST SECTION

The Patient. Let us consider together the first section relating to the patient.

1. *Name of Injured Person.* Write the name legibly in ink, for this is the sole means of identifying the injured person and enables the carrier to connect promptly the report to the proper file.

Age. Too much cannot be said about the importance of giving the age, for any layman knows that a broken ankle in a man of 20 and the same broken bone in a man of 70 may have vastly different consequences. Furthermore, it frequently happens that the age given by a patient to his physician is his true age whereas the age given to his employer may have been different.

Sex. Important oftentimes because the Christian name does not always indicate sex. For illustration, in the South particularly the names of women sometimes are quite masculine, e.g., Johnnie, Johnsie, Willie, etc.

2. *Address: Number and Street.* This is important for the purpose of identifying and locating the employee. Frequently we find that the employee has not registered his latest address with his employer. The address should be complete, giving both the number and street and the name of the city, town and state. Many times in the larger centers there are streets of the same name within a few miles of each other; perhaps we should say in the adjacent suburban communities. If the insurance carrier has not the correct address of the injured man, it may be difficult for it to forward compensation when due.

3. *Name and Address of Employer.* This is most important in identifying the employer of the injured man as records in most insurance companies are filed by the name of the employer. Therefore, this information assists many times in making a prompt investigation. Frequently the medical report is the first notice of an injury, and if the employer's name and address are not given, the report is returned to the physician for completion.

SECOND SECTION

The Accident. 4. *Date of Accident: Hour.* The date of the accident and the definite hour of occurrence are of great importance, as this information helps to determine whether the accident occurred during working hours and during the policy period. In some states, if the accident occurs late during the working period, the compensation period does not start until the following day. In other states it is otherwise. The date of the accident and the hour of the accident are also important because many times the making of a claim alleging an accident is an afterthought on the part of the claimant and it becomes very important to check the statements given to the physician. Many times there is a discrepancy which helps us to determine whether the accident is a legitimate one or not.

Date Disability Began. The date disability began is equally important with the date of the accident because in many instances there are delayed disabilities of perhaps days, weeks or months following the alleged accident. The date of disability is also important, possibly in determining the causal relationship between the alleged accident and the condition found by the physician. This information is becoming increasingly important in view of the widening of the scope of many Acts to include occupational diseases. In such cases we have no date of disability or accident unless it is given by the physician. In occupational diseases it is important to indicate the date the man quits work and the date he actually became disabled. They may be two absolutely different dates. We all recognize that a man may leave work because there is no work for him to do and later on he discovers that he has an occupational condition on account of which he makes claim.

5. *State in Patient's Own Words Where and How Accident Occurred.* It is always valuable and useful to put down in the first person just exactly what the patient said

to the physician as to how the accident happened, e.g.: "I slipped on some water and fell down, landing on my back." Such a statement contains the facts of what caused the fall and the part of the man's anatomy which was injured.

In most instances the physician is the first one to see the claimant following the injury and therefore the claimant is more likely to give an accurate statement then than after talking with relatives and friends. After all, what we are searching for in any investigation is the truth. Many times in compensation hearings the history given to the physician may be the pivotal point on which the decision of the referee or commissioner hinges. It is of great importance to all claim examiners and medical or surgical advisers in determining the question as to whether the condition actually resulted from the alleged injury or not.

THIRD SECTION

The Injury. 6. *Give Accurate Description of Nature and Extent of Injury and State Your Objective Findings.* When we come to this question we are impressed with the fact that this form of report appears to be a great record for a minor injury and a very small record for a large or serious injury. In the latter case the information must be supplemented by a more complete report. Yet it must be always borne in mind that this is a physician's story told to a layman and the avoidance of technical terms so far as possible is much to be desired. However, with a little practice and thought, the description of the injury can be boiled down to a few words, such as "compound comminuted fracture of the right tibia in the mid third," "right inguinal hernia, indirect, scrotal, reducible." If there isn't room on the report, use an extra sheet of paper, for this is one of the most important items on the report. Upon this description depends the evaluation of the case. It also assists greatly in determining the question of whether the disability is being unduly prolonged. The objective findings are of the utmost importance in determining the question of causal relationship between the alleged injury or accident and the condition found by the physician. In a great many instances the objective findings reported by the physician fail absolutely to bear out the diagnosis which he makes. For illustration: we recently read a medical report in which the patient had received an injury. Physical examination described him as entirely normal and described no abnormal or pathologic things about it. Then at the end in his opinion the physician said, "I believe this man is incapacitated from doing any work."

These are the things that bore, bother and annoy.

7. *Will the Injury Result in (a) Permanent Defect (b) Facial or Head Disfigurement? If So, What?* Again a proper answer to these questions is of great importance in determining the value of the case, not only from the standpoint of temporary total disability but from the standpoint of permanent impairment or disfigurement. Furthermore, with the vast experience which the medical departments of insurance companies are having in this highly specialized work, it is quite possible that there may be some treatment which they have learned about through experience in other cases which might lessen or entirely eliminate the disability or improve the end result; and, in connection with question 7, attention is called to the mannequin on the reverse side of the report by the use of which the doctor may indicate the exact point of amputation, injury or disfigurement. Accuracy in making this indication is of great importance because of the difference as respects the provisions of the laws of the various states. It is also important in answering this question to indicate whether there is any restriction and to what extent that restriction extends and where it is located.

8. *Is Accident Above Referred to the Only Cause of Patient's Condition? If Not, State Contributing Causes.* Important because

some states do not recognize the aggravation by an injury of a previously existing systemic condition, compensation in such states being payable only for a disability normally to be expected from that type of injury.

9. *Is Patient Suffering from Any Disease of the Heart, Lungs, Brain, Kidneys, Blood, Vascular System or Any Other Disabling Condition Not Due to This Accident? Give Particulars.* We all know, even a layman, that fractures and injuries generally do not clear up readily if a patient is suffering from syphilis or diabetes. The disability will be prolonged if the patient has an arteriosclerotic condition, kidney trouble or a bad heart, or any one of a number of diseases. It is therefore important that the attending physician, the surgical director and the claim examiner have the fullest information so that they may coöperate together in shortening the period of disability.

10. *Has Patient Any Physical Impairment Due to Previous Accident or Disease? Give Particulars.* This is important, too, in that failure to give an accurate answer might involve the insurance carrier or employer in making a payment for a permanent impairment or amputation which had previously been paid for.

11. *Has Normal Recovery Been Delayed for Any Reason? Give Particulars.* This question is closely related to questions 8 and 9 and affords the physician an opportunity further to explain the reason, if there is such, for the delay in reaching normal recovery.

For illustration: A few years ago it was discovered in New York City that among a certain group of employees of a particular nationality there were many injuries of a definite type for which there seemed to be no logical explanation; neither was there any discoverable reason why recovery was so long delayed. It was learned that these injuries were self-inflicted and self-perpetuated and that the entire racket was being carried on by a well-organized group. This fraud cost the insurance carriers and the employers hundreds of thousands of dollars. Therefore, the importance of this question and the answer thereto.

Again, we want to point out that under "The Injury" questions 6 to 11 inclusive contain pretty nearly all the law and the gospel as respects the problem of determining the actual loss cost of the case and the relation of the condition to the alleged accident.

FOURTH SECTION

Treatment. 12. *Date of Your First Treatment. Who Engaged Your Services?* This is of extreme importance, particularly in cases where treatment has been delayed and possibly an infection has gained the upper hand. Oftentimes this date is important as it may indicate treatment prior to the actual date of this accident. Again, it might involve the question of which of two carriers, there having been a change in carriers by the employer, is responsible for the alleged injury and ensuing disability with consequential expense.

This might be important in a case, as we understand it, of the following type: A man has an ulcer within the eye. The sensation of its breaking through the surface might be the same as a foreign body entering the eye, and in such a case there is a great responsibility upon the doctor to determine whether there is a causal connection between the condition which he finds and the story of the accident as given by the patient.

The second part to this question—"who engaged your services"—is important in many states because of certain provisions of the law.

13. *Describe Treatment Given by You.* It is helpful, in fact essential, for the medical or surgical director to know exactly the kind of treatment rendered by the attending physician in order to determine whether it is proper treatment for the injury or condition, whether it is the most up-to-date treatment or perhaps an outmoded treatment which has been found absolutely useless or almost useless in treatment of the

condition present compared with modern treatment.

Also, if there was a wound, was it sutured? Was it drained? Was it cleaned up?

If there was a hernia, was there an operation?

If there was a fracture, was it reduced? Was it splinted? What was the nature or kind of splint? Was a satisfactory reduction obtained?

14. *Were X-Rays Taken?* Very important in certain types of injuries.

By Whom? This is necessary information in order to determine whether or not an expert roentgenologist interpreted the plates. To one inexpert in the reading of *x*-ray plates a natural suture line in the skull might cause a mistaken belief that it was a fracture line. It would be even more serious if there were an unrecognized fracture.

It makes all the difference in the world what the *x*-ray findings are.

When? Of course, it is important that the date the *x*-rays were taken should appear in the Surgeon's Report.

15. *X-Ray Diagnosis.* We have already referred to the importance of this subject.

16. *Was Patient Treated by Anyone Else? By Whom? When?* Physicians recognize the fact that quite often patients dissatisfied with a diagnosis travel from one doctor to another.

In some states the law requires that the physician in filling out his report cover this question fully. Sometimes an answer to this question would disclose the fact that the patient had been treated for a long time previously for something which he now claims results from a recent accident.

17. *Was Patient Hospitalized? Name and Address of Hospital.* The correct answer to this involves the securing of hospital records, for ultimately there will be a hospital bill and this involves, again, the question of loss expense as represented in reserves.

It is also helpful to determine again whether proper and adequate treatment has been and is being given for the condition described in the report. Investigation sometimes discloses that the injury was not of sufficient severity to require hospitalization. This means unnecessary and additional expense and reflects upon the judgment of the physician.

18. *Date of Admission to Hospital? Date of Discharge?* Important to the insurance carrier and employer in checking up bills received from the hospital. The answer also has the same value as respects the answer to question 12.

19. *Is Further Treatment Needed? For How Long?* This, too, is vitally important in determining reserve figures and also the question of whether specialized treatment which might be recommended by the physician would shorten or eliminate the disability. Very important that we know the period of further treatment if such is recommended.

FIFTH SECTION

Disability. 20. *Patient* {*was* / *will be* *Able to Resume Regular Work On?*

21. *Patient* {*was* / *will be* *Able to Resume Light Work On?* While these two questions may not be the most important on the report, they are equal in importance to any that appear thereon, for upon the answers to these questions depends the date on which compensation should cease. They serve to satisfy the various boards or commissions that the insurance carrier, or employer, is warranted in stopping compensation on that date. The physician should bear in mind that while questions 20 and 21 are tied together, at the same time they are absolutely different questions. Question 20 relates to the fact that the employee was able to resume his regular work and 21 indicates that he was able to resume light work. The answers to these questions are of value to the employee, insurance carrier, employer and the administering board or commission.

22. *If Death Ensued, Give Date.* This question is obviously a very important one for record purposes.

SIXTH SECTION

The last group of questions, particularly "Remarks" will quite likely serve no good purpose if the physician has carefully and thoroughly answered all the foregoing questions. It may be used to elaborate on answers to previous questions or where a physician feels that the injured man is able to leave the hospital and the injured man has a contrary view. Hospitals very often make very satisfactory boarding places to men without homes.

The questions which follow immediately in this section relate to the personal history of the physician and are required in some states and are of value in all states.

Of course, the report should be signed personally by the physician—by no one else. In some states the law requires that the reports be attested as they may be used in evidence.

The information which may be contained in this section of the report is also of great value to the experienced surgical director of a company in assisting young physicians who have recently graduated from medical school and who, obviously, may not have had such experience as to justify the expectation that the end result or end treatment will be in every way as satisfactory as that at the hands of an older and more experienced man. And, again along this same line we would emphasize the desire of all medical departments to have the full coöperation, so far as the consulting facilities are concerned, in all cases which have problems connected with them. Teamwork is doing much in lessening disabilities and obtaining better end results, the aim of all highly professional men and hospitals.

POST-MORTEM EXAMINATIONS

It is generally admitted that post-mortem examinations should be held in all suspected industrial cases. Here again all that is sought is to bring out the facts. Quite recently we had the case of a patient who died suddenly following a leg fracture after three and one-half months' disability. It was the opinion of the attending physician that the patient had died of heart disease. The post-mortem examination, however, disclosed that the patient died of a pulmonary embolus and, therefore, that the death was the result of the original injury.

To the contrary, in another case it was supposed that the patient had died of silicosis and the post-mortem revealed that there was no evidence of silicosis, that the patient had really died of pneumonia.

Such examinations quite often satisfactorily solve all existing doubts as to the cause of death.

CONCLUSION

So, we come to the close of this article with the hope that what we have tried to present will be valuable in improving the type of report furnished by the medical profession to insurance companies in compensation cases. It may promote a better understanding between the two groups, so that, with the explanations given, the physician will no longer feel that the preparation of a medical report is a dull and uninteresting proceeding, but that he is rendering a worthwhile service to a valuable client. Perhaps also, the insurance examiners will no longer be bored by the report in which the physician tries to be both lawyer and doctor; the verbose report in which there are long and elaborate explanations which say nothing; the report which leaves you, when you are through reading it, with no idea of what has happened to the patient, what is the matter with him or what treatment he has received; the report which calls every case of a patient with a lifting back strain a malingerer; the report which is all opinion but does not give the facts upon which the opinion is based.

MEDICAL EXPERTS AS WITNESSES

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BALTIMORE, MARYLAND

IT is a debatable question as to when a witness becomes an "expert witness." Various rules have been laid down by learned text writers to determine this and courts of last resort have been called upon to decide, but their decisions, like most legal adjudications, are not harmonious. In a general way, before one can qualify as an expert, it must appear that he has acquired some special knowledge or skill in certain lines, not generally possessed by the ordinary practitioner. Expertness may come from academic study, or from wide experience, or from the two combined. A reasonably safe working rule would be that where the question calls for the expression of opinion, evidence based on special study or wide experience, "expert" testimony is necessary. It is, of course, not limited to physicians or surgeons. There are "experts" in all lines.

Some years ago there was a celebrated case on trial in the Federal Court in Baltimore, involving litigation by an electric company transmitting electricity to Buffalo. The company engaged Dr. Henry Rowland, then Professor of Physics at Johns Hopkins University, as an expert. Some time after Professor Rowland had given this public utility the benefit of his expert knowledge and experience, differences arose between them as to the value of his expert services. The disagreement not having been amicably adjusted, Professor Rowland sued the electric company for $10,000, his fee as an expert. The great Joseph H. Choate represented the electric company, and the late William L. Marbury, Sr. represented Professor Rowland.

In Mr. Marbury's opening statement to the jury, he characterized Professor Rowland as "perhaps the greatest physicist in America." Choate put on his nose glasses and looked inquiringly at Marbury as if he were covering a great deal of territory. Professor Rowland, as plaintiff, then took the stand, gave his qualifications and the nature of his research, and described the value of his services in the case in which he had been employed. He was then turned over to Mr. Choate for cross examination. Mr. Choate's first question to Professor Rowland was, "Dr. Rowland, who, in your opinion, is the greatest physicist in America?" Without a moment's hesitation, Dr. Rowland answered, "I am."*

Some time later, at a Johns Hopkins dinner, Dr. Gilman, then President of the University, chided Professor Rowland for his lack of modesty on the witness stand. Professor Rowland in reply said that President Gilman failed to take into consideration a very important factor, which was that when he was put upon the spot on that occasion by Mr. Choate, he was under oath to tell the truth, the whole truth, and nothing but the truth. The Federal jury gave him a verdict for $9,000. Most doctors will probably be spared embarrassment of this type, but Professor Rowland was, at any rate, equal to the occasion.

This anecdote perhaps suggests the importance of self possession on the witness stand as a qualification for the expert witness.

Self possession, without previous preparation, will not of itself suffice. There are many other requirements. Learning alone is not enough. Let the expert witness always be mindful of the fact that he is being called on by the courts to make a

* This incident in the trial is authenticated by a letter to me from Mr. William Lee Rawls, a partner in the firm of which Mr. Marbury was the senior member. Mr. Rawls also participated in the trial.

scientific contribution to the cause of justice, in an attempt to solve a disputed problem between litigants. To be genuinely useful, he must be impartial and just. He must avoid partisanship and all appearance of it. He must not be personally interested in the outcome of the litigation. However great the temptation to be enthusiastic over the side of the case on which he is employed, he destroys his usefulness, and brings reproach upon his profession, if his zeal gets the better of his judgment. He must never appear to be reluctant to make an admission which is favorable to the other side, if the facts warrant it. Truth and candor will make the testimony in other respects more likely to be accepted by court or jury. An honest expert is useful to both sides, a dishonest one to neither.

The besetting sin of all experts is that they talk professional jargon to laymen (to judge or jury as the case may be). One must remember what a jury is—a cross section of the community, oftentimes gathered up at random from the highways and byways, from lists of taxpayers or registered voters, white or colored. Jurors are of all nationalities, and may be fishermen, clerks, storekeepers, cobblers or push-cart men. Some of them can hardly read or write, and many speak English most imperfectly. Some are without much understanding. The idle and those on relief are now seeking jury service, for the "per diem" involved, whereas the banker and business man are too often seeking exemption from jury service. Year after year new statutory exemptions are lobbied through the legislatures of the several states to exempt such classes as federal, state and municipal employees, registration and election officials, school teachers and college professors, physicians, surgeons, dentists, ministers, lawyers, and active members of the militia. To the shame of Maryland, we must confess to a most iniquitous provision in our laws whereby anyone may purchase exemption from jury duty by making an annual subscription of ten dollars for the support of the state militia. This law is used largely by bankers, insurance men and business men—our tin soldier jury slackers. The practical result of such exemptions is to leave the State courts with an inferior grade of juror. Hence all litigation which is capable of being removed (by reason of diversity of citizenship) is transferred from the State to the Federal courts. The clerk's offices are suffering from loss of fees and costs. The raw material that is left available for jury duty is not always of the highest caliber. It is that residuum to which the expert speaks when on the witness stand. Hence the importance of getting down to their level and addressing one's self to those men (and women in many states) in such language that they can understand a scientific situation by the use of terminology with which they are more or less familiar.

Conceded that science is oftentimes incapable of translation without the use of scientific terms. In such cases, the scientific description must be used, but only insofar as is absolutely necessary. Then it should immediately be translated, as completely as possible, into the language of the man on the street. In that way knowledge and special skill become helpful. Otherwise, the expert is often a useless and expensive ornament. Parenthetically, I might say that lawyers are just as bad, if not worse, than medical men, in their use of technical terms.

What justification can the medical expert find for the following answer* (taken from an official transcript)?

(Lawyer) "Q. Describe in detail what, if any, tests you made of the plaintiff, and for what purpose these tests were made?

(Doctor) "A. In addition to the usual complete physical exami-

* Medical deposition taken in Baltimore, Maryland September 12, 1938 for Supreme Court of New York in Liggett Drug Store Case.

nation, the patient had a complete blood count and phenolsulfonephthalein test of kidney function, and Mosenthal test of same, and estimation of the non-protein nitrogen and sugar content of the blood flow, and serological test for syphilis and *x*-ray of the kidney and culture of the urine; and urea clearance test, cystoscopic examination, and nose and throat consultation and Addis count and Rubins test."

Other pointers for the medical witness might be:

Avoid flippancy on the witness stand. Your profession is too dignified for jokes on such occasions.

Speak loudly enough to be heard, and slowly enough to be understood. Pause between question and answer until you are sure you understand what is asked, and then answer candidly, even if the answer is "I don't know." It will be refreshing to the jury to find a learned man willing to make such an admission. They may then believe some of the other things which you have said. (Robert M. Morse once asked the longest hypothetical question ever asked, twenty thousand words long, of Dr. Jelley, Boston expert on insanity, in the famous Tuckerman will contest in Suffolk Probate Court. The question concerning the testator's mental condition took over three hours to propound. Strangely enough, the answer comprised just three words, "I don't know."*)

Be not too flamboyant, either in your dress or in your manner of delivery. Be courteous to counsel on both sides. The opposing counsel may not always be courteous to you. The best way to humiliate him for his rudeness is by extreme courtesy on your part. The jury will then be with you, and against him.

Lawyers try many tricks on experts. Their knowledge of medicine or surgery is often very superficial, but they can ask many seemingly simple questions calculated to confound you. They may have read a little medical literature for the occasion. They may have taken the names of late medical books on the particular subject, and then ask you if you are familiar with this or that author. Perhaps you are, perhaps you are not. Note the word "familiar." You may know there is such a work. You may have read certain chapters in it in which you were interested —and yet you may not be "familiar" with the work as a whole. If not, do not hesitate to make it clear. There are hundreds of late law books with which they are not familiar, and of which they have never heard. Some are "familiar" with none—they practice by ear. In a state where examination for admission to the bar was conducted orally in open court, I once heard the son of the then Governor being questioned on international law. He was asked what authors he had read on that subject, and named Grotius and Vattel. The presiding examiner then stroked his long white beard and said, "I suppose you would concede Krupp's Foundry to be a pretty good authority on international law, would you not?" The student replied, "Yes, sir, I have read part of that work, but I cannot say that I am familiar with it as a whole." Whereupon they moved his admission to the Bar.

May I append Woldman's description of the medical expert on the witness stand:

"With erudite profundity,
And subtle cogitabundity,
The medical expert testifies in court:
Explains with ponderosity
And keen profound verbosity
The intricate nature of the plaintiff's tort.

"Discoursing on pathology,
Anatomy, biology,

* Courtesy of Jad's Service, Cleveland, Ohio. (5 Ohio Law Reports 45.) Baltimore Daily Record, September 20, 1938.

Opines the patient's orbit suffered thus:
Contusions of integuments
With ecchymose embellishments,
And bloody extravasation forming pus.

"A state of tumerosity
Producing lacrimosity,
Abrasion of the cuticle severe,
All diagnosed externally,
Although he feared, internally
Sclerotic inflammation might appear.

"The jury sits confused, amazed,
By all this pleonasm dazed,
Unable to conceive a single word,
All awed, they think with bated breaths
The plaintiff died a thousand deaths—
What agony and pain he had endured!

"But then the counsel for defense,
Devoid of garrulous eloquence,
Asked, 'Isn't it true that all you testified
Means merely from a punch or two
The plaintiff's eye was black and blue?'
'Yes, that's correct,' the doctor meekly sighed."*

* "The Expert Witness," by Albert A. Woldman, *Docket*, January 1933. (West Publishing Co., St. Paul, Minn.)

No single method of reduction is suitable for all types of epiphyseal displacement. When manipulative correction is employed, it must be adapted to the needs of the individual case.

THE EXAMINATION OF A PATIENT FOR MEDICOLEGAL PURPOSES

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THE main object of all medical endeavor is to restore people who are sick or injured to as near a normal condition of health and ability to carry on their gainful occupations as is possible. This is of great importance in the economic phase of medical treatment, particularly in personal injury cases. A large percentage of people are either interested in or employed by some branch of industry. All industries have both physical and mental hazards. Accidents and disabilities, and claimed accidents and disabilities, arise in great numbers daily. Vast numbers of cases are continually coming to various courts and commissions for the adjustment of claims and the determination of loss of earning power, but there is great and intolerable variance in these claims. Unfortunate and unreasonable disputes arise as to the diagnoses, causes of conditions or claimed conditions, and these produce heated arguments before tribunals of adjustment and arbitration.

Many patients and claimants are poor. As can be readily seen, this introduces an important factor involving the question of money. The moral sense and administration of justice and fairness is always, by all odds, best for all parties concerned. It is a sine qua non that the best and most expert treatment that can be given an injured patient is for the greatest amount of good for all who are interested in the case.

It certainly is not fair for a claimant to continue to draw compensation at the rate of $18.00 per week under the impression that he is not able to work when as a matter of fact he is able to carry on his usual occupation and earn $35.00 per week—which he and his dependents need. This unfairly produces chronic invalids and unjustly deprives a wage earner of the opportunity to accomplish the things he reasonably should. To induce protracted disabilities is to produce chronic invalids and neurotics. On the other hand, it is not fair for a claimant, suffering from a disability, the result of an accident, and who is not able to carry on his usual work, to be denied monetary compensation which is rightfully due him.

The theory of compensation for injury is not new. In the Old Testament it is stated,

"And if men strive together, and one smite another with a stone, or with his fist, and he die not, but keepeth his bed:

"If he rise again, and walk abroad upon his staff, then shall he that smote him be quit:

"Only he shall pay for the loss of his time, and shall cause him to be thoroughly healed." (Exodus 21:18–19)

It should not be left to chance what particular medical man should treat certain cases. Industrial surgery is a special branch and should be delegated to those particularly skilled in this type of work. A doctor chosing to do industrial work should educate himself along these lines. He should have a thorough understanding of compensation laws, and should coöperate with all interested parties to the fullest and fairest extent. His duty is to report promptly to them his findings on examination and his honest opinion of the case. The best work is secured today under conditions whereby medical men are unhampered by the continuous disturbance of economic competition and the frequently necessary struggle for existence.

It is best for the employee and the employer for an experienced and capable surgeon to be paid $100.00 to repair an injured part of the body and secure a result with a disability amounting to $500.00

than to pay a doctor who is not skilled in the handling of such cases $50.00 and secure a result where the disability will be $2,000.00.

The advancement of science and the increase of medical ability has reached such a stage that there should no longer be a wide divergence of opinion as to the extent and causes of disabilities. Certainly, if competent doctors for both sides meet and consult and examine the case, one will not estimate a disability at 75 per cent while the other estimates the same disability at 15 per cent. I suggest that competent physicians for both sides examine the case in conference, and without predjudice, before the case is presented to any tribunal. If such procedures were followed, the greatest percentage of cases would be settled without trials or contests.

When competent doctors examine patients in private practice, the patients are carefully studied, the various angles are impartially discussed, the questions of diagnosis and treatment are considered over a period of time, particularly if there are any points of difference. After these matters have been considered, after the physicians have read in standard works concerning the various questions, and after careful thought has been given about the best interests of the sick person it is not often that there is any great difference of opinion. The reason for this is that the chief interest is in the diagnosis and treatment and is not influenced by any court procedure nor by anything except scientific medicine and the welfare of the patient. Certainly, after fair consideration, this should be the case in arriving at the diagnosis and disability in any injury or alleged injury. The primary purpose of a medical examination is to determine the facts as based upon a study of the case and should not be influenced by any other thought. Frequently the interests of an injured person are better served by competent and fair consultations. Such a procedure as this will go a long way in increasing the weight and credence placed upon the testimony of doctors as given in courts.

The jury, judges and commissioners arrive at a conclusion concerning the merits of a case by considering factors such as pain, loss of function, deformities, and loss of earning capacity as well as factors of legal merit. It is necessary, therefore, to present the actual and the complete examination findings, without exaggeration and without underestimating the disabilities.

Of course, there is only one way to prepare a medical case for legal determination and that is to make an intelligent and sufficient examination. This includes not only a physical but also a mental study. To make such a survey successfully it is necessary, in the first place, to obtain the confidence of the patient by fair means and by a manifestation of interest. This study should be complete and thorough, and should estimate, with fairness, the value and weight to be given on the claims.

It may safely be stated that the most careful and skilful examination of the patient is for the best interest of the plaintiff and defendant alike.

A complete diagnostic survey is necessary in practically all cases which may result in a trial before some court of record. It is not sufficient to stop with an examination which discloses absent knee jerks, a Rhomberg, and lack of coördination. Certainly, in such a case, a Wassermann test should be made, the pupillary reflexes should be determined, and a complete neurologic examination carried out. If a patient has suffered an injury and complains of paresthesia of the lower extremities, ataxia, altered vibratory and position sense, and a paraplegia, and evidence of injury to the pyramidal tracts, the examination should not stop at this place. A skilful physician will usually find, on making a careful blood examination, that pernicious anemia is the cause of the disability and that it is not due to lifting on a joint of pipe. Extreme nervousness and trembling and general debility may be

found, after a careful examination, to be due to multiple sclerosis. These are only a few examples showing the necessity for making a complete examination in any case that is of any consequence. Such an examination may prevent a doctor from being humiliated and discredited in court, when cross examined by a shrewd lawyer, well informed on the particular case.

A speaker on any subject must keep the kind and class of his audience in mind. Different classes of audiences demand different explanations. When any matter is presented, one must consider the kind of people to whom it is presented in order that an explanation which can be understood will be made. Otherwise the issue is wasted, time is lost, and the case is not understood.

Juries are not composed of doctors, nor of men familiar with technical medical terms. Judges, commissioners and lawyers are not doctors and hence are not presumed to be familiar with complicated medical language. In preparing a case to present the facts for study to others than doctors, understandable terms and words must be used. Jurors know what one is talking about when the words, "wrist" and "ankle," are used, but ordinarily they do not know what is being discussed when such terms as "trapezium," "scaphoid," "astragalus," or "os calcis" are used.

The explanation of the word, and significance of the term "fracture" must be made clear to laymen. Fractures of the transverse processes of the spinal vertebrae are frequent and they usually heal by fibrous union and do not show bony union in the *x*-ray pictures. Such patients practically always recover and are able to carry on their work and have no disability. These are facts established by many hundreds of cases. Certainly it is not wise to elaborate about the patient with a broken back and make all the implications that are carried with such statements without some fair explanation of the particular case in point so that it may be reasonably understood.

The anatomic explanation that the spinal cord ends at the second lumbar vertebra will throw some light on the frequent claim that a slight compression fracture of the fourth or fifth lumbar vertebra is pressing on the spinal cord. The statement that a patient has a broken back, and the allusion that he has an injured spinal cord must be carefully explained to the attorney with reference to its significance as to disability.

One of the most frequent types of claims is for injury of and disability to the back. A list of the causes of backache would include practically every disease, injury and congenital deformity that exists. Due to the frequency of these claims, there are a few commonly accepted points which have been proved by experience and which are based upon anatomic findings with which an attorney should be familiar. A history of a gradual onset of pain which becomes progressively worse is most frequently due to an arthritis or to some disease. The patient should be required definitely to locate the pain and this location should be noted. At subsequent examinations it should be determined if this location has changed. Pain in the back due to a genuine injury or a really definite cause does not switch and jump around all over the back. It is well known that a patient who has a pain in his back which is the result of a sprain of the muscles or ligaments has more trouble and more disability in the morning, after a night's rest, than he does with moderate exercise.

After it has been explained to a layman, he is able to note some of the physical characteristics of the patient: his build, the presence or absence of abnormal curves in the spine, whether or not there is tilting of the pelvis, and other representative findings. Always examine for the presence of muscle spasm or atrophy in the region of the area complained of. The motions of the spine are easily obtained and can be seen by any layman. The patient should be requested to bend to the right and to the left, to bend forward and to bend backward. The location of the pain during these move-

ments should be determined. Be particular to ask if the pain is increased or relieved by any one motion. In genuine back pain due to injury, the pain is increased by some motions and relieved by others. In a real case of injury, the patient does not complain of severe pain when all the various motions of the back are performed. In flexing the back, tension is placed on certain muscles which may be painful on account of an injury, but when extension is performed, these muscles are relaxed and the pain is relieved.

The significance of valuable laboratory tests will be lost unless a careful and clear explanation of their meaning is made to laymen.

One of the most important points to be brought out during the examination is the history and description of any previous illness.

Occupation is a matter to be considered because certain occupations place a definite strain on certain muscles and this strain is usually a continuous one.

An effort should also be made to estimate and determine the type and characteristics of the individual. Different types of people react differently to the same amount of pain. It is necessary to estimate whether the patient is excitable or nervous, or is of a phlegmatic disposition.

In examining the extremities, measurements can be taken with the tape measure with the expenditure of very little time. Such measurements are very impressive to laymen in determining definite conditions.

The subject of fractures of the skull frequently presents itself. It cannot be too thoroughly emphasized to laymen that the skull fracture is not the thing of importance, but that it is the damage to the contents within the skull that is of importance. Always make the more common neurologic tests, which require not skill so much as time. Determine the condition of the deep tendon reflexes such as the knee jerks, ankle jerks, biceps and triceps reflexes. Make the usual tests for sensory disturbances, such as the distinction between sharp and dull, the ability to determine pain and touch, the vibratory sense and the sense of localization. The significance of the various common findings can, with intelligence, be explained to someone not versed in medicine in such a way that they will be understood. If anything of an abnormal nature is found, a careful and more exhaustive examination of the nervous system should, of course, be made.

Injuries to the skull and spine have so frequently been considered by the layman as serious, dangerous and lasting disabilities and it has been so repeatedly emphasized that a person with a broken back will never be able to do full work again that a fair and honest explanation of the terms and meaning of the particular findings in the case should be made.

Especially in medicolegal cases is it wise to make repeated examinations and observe the patient over a period of time. Well do I remember taking care of a man who had compression fractures of the third and fourth lumbar vertebrae—and they were compressed to an extreme degree. No neurologic changes from the normal were present, but definite muscle spasm was found. When I dismissed him from my care about nine or ten weeks following the injury, I estimated, in good faith, his partial permanent disability at 65 per cent. About two years later, this same man, with the same compression fractures, came to my office with the statement that he had no disability, that he had no pain, that he felt himself fit in every way to resume any kind of manual labor. A careful physical examination revealed no objective signs of disability, and I felt that there was no reason why he could not carry on the same work as before the injury. The moral of this experience is that in preparing cases for legal presentation, we must impress the fact upon laymen that time is a great healer and that repeated examinations and observations are frequently necessary for the proper evaluation of the disability in such cases.

It must be explained that *x*-rays do not determine disability nor lack of disability.

X-ray pictures of a fracture that looks terrible may be *x*-ray pictures of an extremity that has excellent function. Again, of course, the reverse may hold: an *x*-ray picture may look good, but function of the extremity may be impaired.

To determine the presence of an ulnar nerve injury, observe whether the patient can abduct or adduct his fingers. Abduction and adduction are not possible where the ulnar nerve has been injured, for the reason that the ulnar nerve supplies the muscles which control these motions. Similar tests, based on anatomy and sound reasoning—tests which are obvious—may be used in different types of cases.

Legal cases, involving medical questions, are prepared by doctors and lawyers together and the conditions must be explained in such a way that the reasons are evident to lay minds. Attorneys must have familiarity with the case in order that they may present the matter in an intelligent manner to laymen in order to assist them in reaching logical and reasonable conclusions.

Most objective findings are overlooked because not enough time is taken in determining the common conditions and examining for the findings which may be obvious. One should always present all of the facts to the lawyer consulting you, giving him both sides of the case. This is the only fair way and the method necessary to prevent many court surprises and difficult situations. It is advisable, wherever possible, to have the attorney present when examinations are made. Observation is one of the best ways to become familiar with a case, and in this way the attorney has the facts that are important indelibly impressed upon his mind in such a fashion that he will remember them and they will have a meaning.

The question of inguinal hernia due to an injury frequently arises. Of course, we know that an inguinal hernia, if it is ever traumatic, is extremely rare, but when such a case presents itself, a complete history is essential. It should be considered whether or not the cause could reasonably have any connection with the condition claimed. Other important anatomic and medical ideas and reasons should be carefully discussed with the attorney in connection with such a case.

No matter what type of disability or injury is to be tried before a court of record, the points should be explained to the attorney in language which he will understand. Reasons for such explanations are given so that the attorney can, in turn, discuss the matter intelligently with the person or persons passing upon the merits of the case.

The attorney must understand that certain diseases produce findings that are visible to an intelligent observer. A patient with a definite sciatica walks with a list to one side, and assumes a quiet position without continuous twisting and squirming and jumping around in his chair.

In the preparation of a case for legal presentation, if the facts are carefully obtained, without prejudice and presented in terms that can be understood by lawyers, courts and juries, the most important steps in securing an equitable adjustment will have been obtained. It again must be repeated that time and patience are necessary for the proper and fair preparation and presentation of such technical matters to non-medical men.

The record of injury, its time, the dates of treatment, the various complaints, the progress notes and the findings must all be kept in writing because the doctor will almost certainly be called upon to testify concerning these facts. A doctor handling a large number of cases will not make a proper impression if he relies upon his memory as to a large number of complaints, details and dates.

A great part of the reward in the practice of medicine is the pleasure derived from studying and examining cases and discussing them with competent colleagues. This is one of the ways in which we keep up with the advances in medicine. It is best for the patient, and certainly is the most efficient way to prepare a case which is to come up before some tribunal for a hearing on the facts and an estimation of the disability.

CONSERVATIVE TREATMENT OF CEREBROCRANIAL INJURIES

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SINCE conservative treatment may be applied in probably 90 per cent of cerebrocranial injuries, about 10 per cent only being surgical, identification of the types of injuries and the pathologic background of each is necessary in determining what steps should be taken. The problems to be faced vary, of course, in a long series, but whether the injury be mild, moderately severe, or severe, the treatment changes as time elapses. Consequently, prompt consideration of the change in symptoms should always indicate the immediate procedure.

Our treatment, except in those cases in which a change from the normally expected recovery requires operative measures, is based upon the consideration of four important pathologic factors: (1) shock, (2) cerebral hemorrhage, (3) edema, and (4) brain damage. Shock is a general feature; the other three have to do with intracranial lesions. Omitting any detailed discussion of the pathology and causes of shock[1] and of the diagnosis and operative procedure in massive intracranial hemorrhages, we shall outline our procedure in accord with the change of symptoms as time elapses after the injury. Problems dealing with the control of shock, hemorrhage, edema, and brain damage and with the use of roentgenography, sedatives, and spinal drainages are those to be considered in the conservative treatment of cerebrocranial injuries.

THE IMMEDIATE PROBLEM

As soon as the patient comes under his care, a surgeon must ascertain enough of the nature of the injury to determine the possibility of an immediate fatal termination. Much time can easily be wasted at this period in unnecessary examinations and much harm done by unnecessary movement of injured parts. The question is—"Is this patient in danger of immediate death?" Patients die from shock, hemorrhage, and loss of cerebral function. These three things may be easily recognized and their severity immediately classifies the case. Shock is by far the most frequent and at the same time the most favorable factor one may encounter; hemorrhage may be fatal at once, may gradually become an operative problem, or may respond to more prolonged treatment; edema and brain damage usually result in an unconscious state. Although later treatment differs in the case of each of these three conditions, only a simple examination, sufficient to determine whether immediate danger is present should be made at this time.

Shock can be determined by means of blood pressure and, if convenient, by hemoconcentration tests, as red blood cell count and hemoglobin estimation. If the systolic pressure is at or below 50 and the pulse pressure at or above 50, the patient is in shock and must be treated accordingly at once. Blood pressure alone will usually tell whether or not shock is present.

Hemorrhage is often hard to recognize since a massive one will cause severe shock. Prolonged shock plus hemorrhage will prove fatal in almost every instance. A moderate brain hemorrhage will not be serious at once, however, and so can be attended to later. Bleeding from the mouth, nose, ears, or scalp is significant and helps to determine the severity of the injury.

Cerebral edema is the physiologic reaction to trauma and, therefore, an early development after an injury. Its presence

is indicated by (1) a slowing of the pulse and (2) an increase in the pulse pressure.

Brain damage is, of course, the most serious of the four but can be checked up later. The immediate problem is whether the traumatized area contains brain tissue and to what extent. The treatment of the last three conditions—hemorrhage, edema and brain damage—may well be considered together. Rarely can hemorrhage and brain damage be prevented nor can their extent be controlled. Edema, however, may be both prevented and controlled if those measures which are used in the treatment of shock are continued into the "post shock" period.

These first observations having been made without any unnecessary movements of the patient, a brief neurologic examination can be carried out to ascertain the presence of (1) injury to the spinal cord, and (2) injury to other organs and viscera. Spinal cord injury is difficult to detect where unconsciousness due to brain injury has occurred, but usually one can ascertain whether or not complete loss of function is present. Unnecessary movements of the spinal column, however, should be prevented at least until this point can be cleared.

Injury to other organs and viscera may not be determined until after shock treatments have been instituted and their results obtained. Good response to the latter may be considered as a favorable sign of the absence of abdominal organ injury.

Having established the presence of shock, hemorrhage, edema, or brain damage and the immediate possibility of death, one proceeds to treat the condition most prominently indicated. Mild cases of unconsciousness, possibly associated with alcohol and/or concussion, will not show any of the signs of shock, hemorrhage, edema, or brain damage. These patients, if put to bed, will be near a normal condition within ten or twelve hours. A spinal puncture before discharge will (1) establish the presence or absence of mild damage to the brain (blood in the spinal fluid) and (2) prevent secondary edema in alcoholics. Mild cases can be discharged within twelve to twenty-four hours.

CONTROL OF SHOCK

The general rules we have adopted for the first few hours of treatment of shock are based upon the presence of the following symptoms: (1) unconsciousness; (2) decreased temperature; (3) cold and clammy extremities; (4) rapid, soft pulse; (5) rapid, shallow respirations; (6) dilated pupils; (7) decreased blood pressure, both diastolic and systolic; (8) increased pulse pressure; and (9) hemoconcentration (hemoglobin and red blood cells increased).

Note. Any or all except the blood pressure changes and signs of hemoconcentration may be absent, depending upon the general condition of the patient. A high blood pressure existing before the accident may both confuse the physical picture and mask the findings. The face may appear flushed and the body seem warm. A preëxisting cardiac lesion also may change the appearance of the entire picture. These factors may not be ascertained, however, until later examinations and the acquisition of additional personal history.

Rules for Immediate Treatment of Shock.

1. Put the patient to bed; remove shoes; loosen collars, belt, corset or other garments which may cause constriction about the neck, chest or abdomen.
2. Wrap the patient in warm blankets with hot water bottles outside the blankets.
3. Place the patient on his abdomen or side with the foot of the bed elevated twelve inches (more if he is unconscious).
4. Apply antiseptic or clean dressings to any wound. Stop scalp hemorrhage with pressure bandage. As the patient recovers from shock, shave and cleanse the scalp, apply sutures if necessary, and dress the wounds more completely.
5. Prevent loss of body fluid from the skin (sweating) by the use of atropin to close the capillary network; from the alimentary tract by intravenous adminis-

tration of all fluid rather than by mouth or rectum.

6. Keep the fluid volume of the blood at a normal level by the intravenous injection of hypertonic glucose solution 50 c.c., 50 per cent, repeated every half hour if necsssary.

7. Attend to the toilet of the ear canals when hemorrhage is present by merely packing the canal lightly with cotton. *Do not irrigate.*

8. Dull the patient's sensorium by the use of injections of phenobarbital (intramuscularly), amytal (intravenously), chloral hydrate and/or bromides (rectally) and in some cases morphine (subcutaneously) in small doses.

9. Use transfusion of whole blood, 500 c.c. of 6 per cent solution of acacia in normal saline solution.

Note. In the application of these rules one of the important measures to be performed in the treatment of a patient is to elevate the foot of the bed sharply so as to produce a hydrostatic increase in blood supply to the cerebral centers and heart and to prevent the inhalation of the profuse upper respiratory mucus discharge. Frequently over a pint of mucus will be secreted from the upper respiratory tract in twenty-four hours. Before we routinely adopted the use of this position, we observed a fatal outcome for many patients who had survived intracranial damage, but could not overcome the central pneumonia which resulted from inhalation of infected mucus or vomitus. It is our custom to keep the patient over on his abdomen most of the time during the period of shock and during the first six to twelve hours of unconsciousness when he is apt to vomit. A firm rubber-covered pillow is kept under the shoulder towards which the face is turned and the hip is flexed on that side. The elevation of the foot of the bed should be enough to produce dependent drainage. If this proves insufficient, a rubber catheter connected with a sucker may be used to clean out the pharynx. A metal airway may make breathing easier. It is usually easier to restrain excited patients in this position. As the prolongation of shock symptoms may be due to anoxemia, and this in turn may be aggravated by plugged up bronchioles due to mucus or blood inhaled or to edema of the lungs due to exposure to cold while awaiting treatment, dependent drainage is absolutely essential.

The above rules may be altered as the occasion arises but will be found to be quite sufficient to control most of the cases of shock.

Rules for Later Treatment of Shock. 1. Place side boards on the bed.

2. Restrain one or both hands.

3. If the patient has been drinking alcoholic beverages or is nauseated or is bleeding from the nose or mouth, elevate the foot of the bed. Keep him three-quarters over on his face, first on one side then on the other, changing every few hours until he is able to keep his throat clear.

4. After the above symptoms have passed, turn the patient on his back using a back rest or moderate Fowler's position. Turn him frequently.

5. Catheterize, especially if the patient has been drinking alcoholic beverages or receiving hypertonic solutions intravenously.

6. Do not allow a patient to go more than forty-eight hours without nasal tube feeding of high caloric liquids (salt, sugar, egg, cream, etc.) 250 to 500 c.c. two to three times a day.

7. Watching the urine output, regulate acidity of urine and specific gravity and amount of urine.

8. Make a daily examination of the chest, watching carefully for pulmonary edema, hypostatic pneumonia or central pneumonia in order to anticipate trouble with effective treatment.

9. Take a specimen for blood count and blood chemistry for signs of hemoconcentration.

10. Make a spinal puncture if there is no contraindication.

11. Dehydrate, if indicated, by nasal tube and rectal magnesium or sodium sulfate, combined with hypertonic intravenous solutions.

12. Make frequent ophthalmoscopic and neurologic examinations.

13. Examine carefully for other injuries.

Note. Application of these rules will be found sufficient for most cases of shock even though complicated by a prolonged period of unconsciousness. Should the symptoms not improve and further complications appear, one must consider the possibility of intracranial hemorrhage, viscous edema, or severe brain injury.

CONTROL OF INTRACRANIAL HEMORRHAGE, EDEMA, AND BRAIN DAMAGE

Diagnosis of Symptoms. Hemorrhages may be (1) extradural, (2) subdural, (3) subarachnoid, (4) cortical, (5) subcortical, or (6) ventricular. The signs of all are more or less the same, each case being determined by the amount of hemorrhage present. The lucid interval (unconsciousness, recovery, then slowly developing stupor) when present, is very spectacular but not always diagnostic of hemorrhage. Changes in the pupils, such as inequality, contraction, or dilatation, also are significant of a possible massive hemorrhage, but not always a reliable sign. These changes may be due to concussion with minute hemorrhages within one or both third nerve nuclei which lie just outside the opening of the aqueduct of Sylvius.[2] Convulsions of the Jacksonian type may be due to hemorrhage, brain damage, localized collections of fluid, or all three conditions. Increasing hemiplegias are quite similar in their significance and may also be due to any of the above three causes. These symptoms occur when hemorrhage, viscous edema, or brain damage complicates the recovery from shock.

Diagnostic Tests. 1. Ophthalmoscopic examinations. Size and reaction of the pupils, evidence of edema about the nerve heads, changes in the caliber and tortuosity of the retinal vessels, small retinal hemorrhages, and muscular paresis all indicate intracranial complications.

2. Neurologic examination. Positive pyramidal tract changes, sensory responses retarded, changes in motor reflexes show a progressive character of the intracranial lesion.

3. Roentgenograms.

4. Spinal puncture and drainage.

Treatment of Intracranial Hemorrhage, Edema, and Brain Damage. All three of these conditions require limitation of fluids to 1,500 c.c. daily and regulation of fluid balance. This is brought about by:

1. Control of water balance by limiting the fluid intake and regulating the diet.

2. Careful check of the water balance by (a) specific gravity of urine, (b) blood pressure, and (c) pulse pressure.

3. Use of spinal puncture and drainage.

4. Charting the progress of the patient.

5. Control of pain, restlessness or even mania by proper medication.

6. Use of roentgenograms.

Note. Hemorrhage of a moderate degree is best controlled by spinal drainage. Only the more severe cases, middle meningeal hemorrhage and subdural hemorrhages, require surgery. Brain damage may be mild in character and require only rest and time to effect a cure. When damage is severe, a long period of unconsciousness is always present, and treatment should be directed toward the "unconscious state." The treatment, which has been described under shock, must be continued. Since the important thing after shock symptoms have subsided is to ascertain the degree of damage which the injury has caused to the skull, blood vessels and brain, roentgenograms are necessary. These may be taken either before spinal puncture or after a general physical examination, including the initial spinal puncture, has been made.

USE OF ROENTGENOGRAMS

Except in the very rarest cases, roentgenograms should be delayed until the patient has recovered from shock. The only reason for *x*-ray before this stage is that it will furnish information for an immediate operation. Roentgenograms are important (1) to ascertain the presence of foreign bodies, fragments of bone, and involvement of sinuses; (2) occasionally to differentiate between head injury and other lesions; and (3) to use as medicolegal evidence. They

usually should be taken after shock symptoms have cleared, although depressed skull fractures and spinal cord complications require immediate roentgenograms unless shock symptoms are extreme. The operations that must be done at this time, where a roentgenogram would help, are for progressive extradural hemorrhage, compound comminuted fracture, and foreign bodies.

The best roentgenograms are taken after shock symptoms have been relieved and the patient is quiet. Then a clear film may be obtained which will be of real value. The character of the film is important. A flat anteroposterior exposure will reveal the occipital and lateral fractures. A flat postero-anterior (face to the film) reveals fractures of the facial bones, sinuses and temporal depressions. Lateral stereoscopic views, right and left, reveal linear vault and lateral fractures, depressed frontal and occipital lesions. Oblique positions reveal basilar fractures and involvement of the mastoid and petron.

SPINAL PUNCTURE AND DRAINAGE

If *x*-rays fail to reveal any operative indication, then we institute spinal drainage. We believe that whenever the initial spinal puncture (a diagnostic procedure during which 5 to 10 c.c. of cerebrospinal fluid are removed) shows free blood in the spinal fluid pathways, spinal drainage should be done. "Spinal drainage" implies the removal of from 30 to 60 c.c. of spinal fluid, the lowering of the pressure one-half if it exceeds or is double the normal pressure, or the lowering one-third if it is more than double the normal pressure.

Note. Spinal puncture should never be done without making a manometric reading the moment the cerebrospinal fluid shows in the needle. The different gauge needles and the presence or absence of blood will influence the rate of flow so that a visual estimation is often incorrect and therefore valueless.

Spinal puncture and drainage are contraindicated during the period of shock because they increase an already serious condition while a few hours later they may be done with safety.

Another contraindication for spinal drainage is the leakage of clear or bloody cerebrospinal fluid in the presence of a compound fracture of the skull. Under both the above conditions careful observation and experience will inform the examiner whether the intracranial hemorrhage is progressive. Although a spinal puncture will do no harm if 1 or 2 c.c. are removed, there should be only a very rare need for such a puncture.

Spinal Puncture. The function of spinal puncture is threefold: (1) it determines, from the amount of bleeding and from the history and clinical signs, the relative amount of brain damage; (2) it determines the amount of edema of brain as shown by the increase in intracranial pressure not accounted for by hemorrhage; and (3) it is of value as medicolegal evidence. The presence of fractures as shown by roentgenograms is often disputed, but the presence of blood in cerebrospinal fluid is reliable evidence of intracranial damage and makes more understandable the complaints of permanent disability.

Spinal Drainage. To remove unhemolyzed blood from the meningeal spaces and to relieve intracranial pressure spinal drainage is necessary. It should be repeated if there is unhemolyzed blood in the previous specimen, if the clinical signs indicate an increase in cerebrospinal pressure, or if the last manometric reading was 50 per cent or more above normal. It is also important to do a Queckenstedt test since if a patient strains or holds his breath, or if a needle point is against a nerve root or bit of arachnoid, a false record is made.

THE USE OF MORPHINE AND OTHER SEDATIVES

One of the first important decisions that the attending physician is called upon to make is what sedatives should be used to control pain, restlessness, or even mania. Usually the more definite the need for sedation the more imperative is administration by hypodermic injection only. Most head

injury patients not only are nauseated and vomit easily but their intestinal mobility and absorption are decreased. Also a patient who is restless and confused will frequently expel rectal administration, especially if the latter is of large volume or slightly irritating.

Of the injectable sedatives the most common are barbiturates and narcotics. The subcutaneous injection of chloral hydrate in large enough doses to be effective is dangerous, because the effect is toxic and also continues too long. The subcutaneous injection of paraldehyde is unwise because serious infection may develop. Slow action and slow elimination barbiturates such as phenobarbital have little effect if given in small doses, and, because of slow elimination, a too lasting effect if given in large doses. Rapid action, quickly eliminated barbiturate preparations, such as amytal, increase confusion and restlessness when administered in adequate doses. Thus of the barbiturate group, effects of some last so long as to mask the further progress of the patient's condition; those of others have the disadvantage of actually increasing the symptoms for which they are given. These narcotics, if given in adequate dosage, produce symptoms practically identical to those of cerebral concussion with intracranial pressure, and mask the development of a serious condition, such as a middle meningeal hemorrhage.

Additional danger is associated with the use of large doses of narcotics in the very young and very elderly. The use of morphine in adequate doses may produce what amounts to paralytic ileus in some patients, particularly in those who are habitually constipated, whose injuries prevent them from giving force to defecation or who are unconscious. To escape the undesirable effect of using either barbiturates or narcotics alone, we have combined them in cases of severe post-traumatic psychoses. The choice of which drug to use is less important than how to administer the individual drugs. We follow these methods: (1) luminal intramuscularly; (2) amytal intravenously; (3) chloral hydrate and bromides or paraldehyde rectally; or (4) morphine, codeine, subcutaneously.

USE OF CHARTING

The ordinary hospital graphic chart can be used, but since Fay published his head injury chart, the value of the special chart has been established. We have modified the one outlined by Fay[3] in only minor details and believe it is of material benefit in watching the progress of a case.

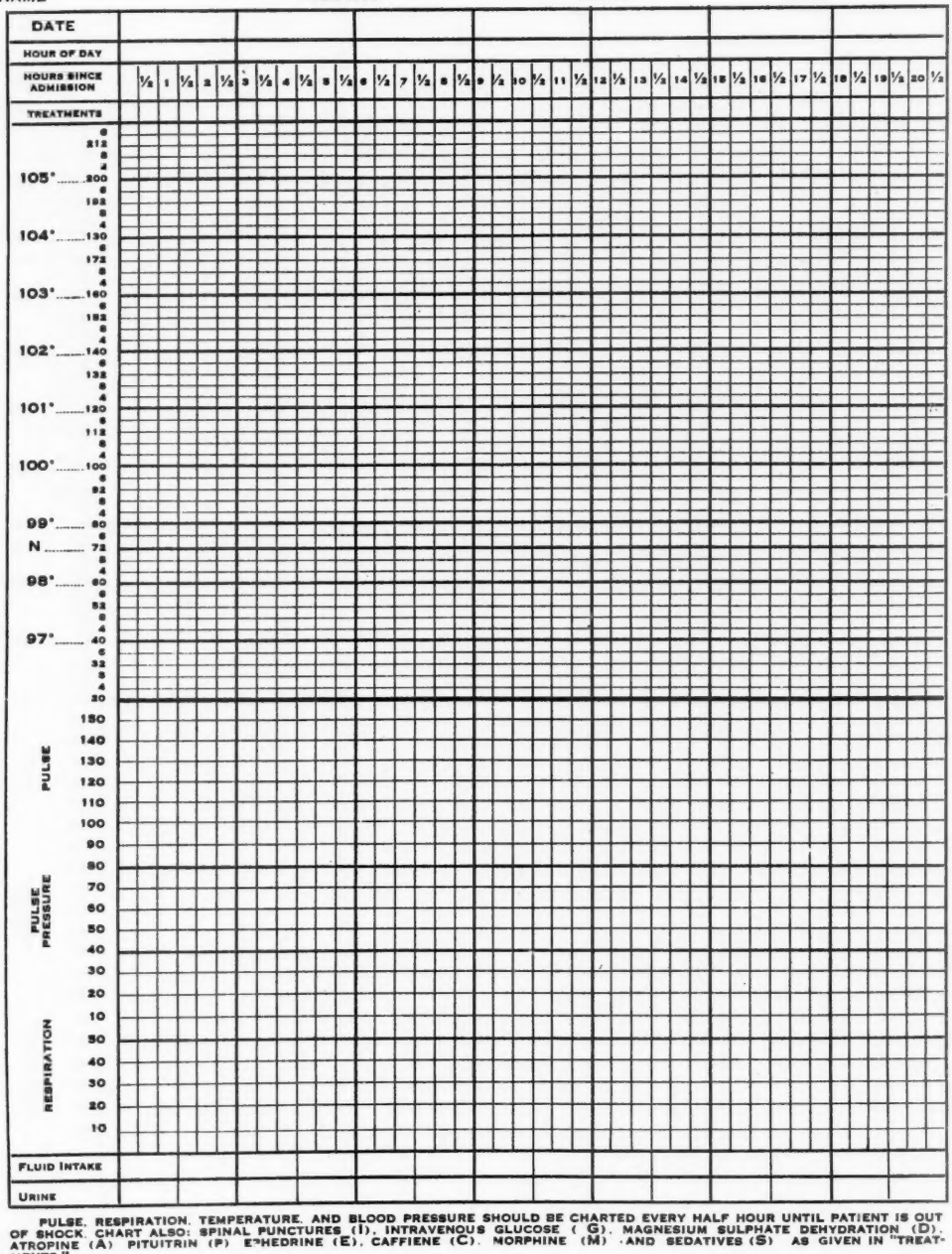
Since the most reliable signs of shock, increased intracranial pressure, and damage to vital centers of the brain are obtained by observation of the condition of the vascular system, notations of the rapid systemic changes are profitably made as often as every half hour in extremely critical cases. Two graphic charts should be used, one for notations every half hour, and the other for the usual four, eight, and twelve hour notes. Space is provided for recording systolic and diastolic blood pressure, pulse pressure, treatments, and fluid intake and output, as well as conventional pulse, temperature, and respiration. Treatment usually can best be guided by the chart, for the psychologic variation is the key to the therapeutic measure.

Moreover, since the surgeon cannot be in constant attendance upon the patient, this graphic chart, upon which frequent notations are made, often gives him a better guide to the progress of the patient then do the observations of changing nurses and resident staff. The permanent record which it affords obviates the necessity of depending upon memory to compare changes in the patient's condition, and the half hour notations tend to increase interested observation of the patient by the hospital nursing and resident staff. A very real advantage is obtained since the time necessary to make the frequent blood pressure, pulse, respiration, and temperature observations requires that close attention be paid to the patient's condition. Such observation may detect a progressive middle meningeal hemorrhage, for example,

THE NEURO SURGICAL CLINIC
SEATTLE

GRAPH—A

NAME SERVICE WARD CASE


DATE
HOUR OF DAY
HOURS SINCE ADMISSION
½ 1 ½ 2 ½ 3 ½ 4 ½ 5 ½ 6 ½ 7 ½ 8 ½ 9 ½ 10 ½ 11 ½ 12 ½ 13 ½ 14 ½ 15 ½ 16 ½ 17 ½ 18 ½ 19 ½ 20 ½
TREATMENTS
105° 104° 103° 102° 101° 100° 99° N 98° 97°
PULSE
PULSE PRESSURE
RESPIRATION
FLUID INTAKE
URINE

PULSE, RESPIRATION, TEMPERATURE, AND BLOOD PRESSURE SHOULD BE CHARTED EVERY HALF HOUR UNTIL PATIENT IS OUT OF SHOCK. CHART ALSO: SPINAL PUNCTURES (I), INTRAVENOUS GLUCOSE (G), MAGNESIUM SULPHATE DEHYDRATION (D), ATROPINE (A) PITUITRIN (P) EPHEDRINE (E), CAFFIENE (C), MORPHINE (M) AND SEDATIVES (S) AS GIVEN IN "TREATMENTS"

CHART I.

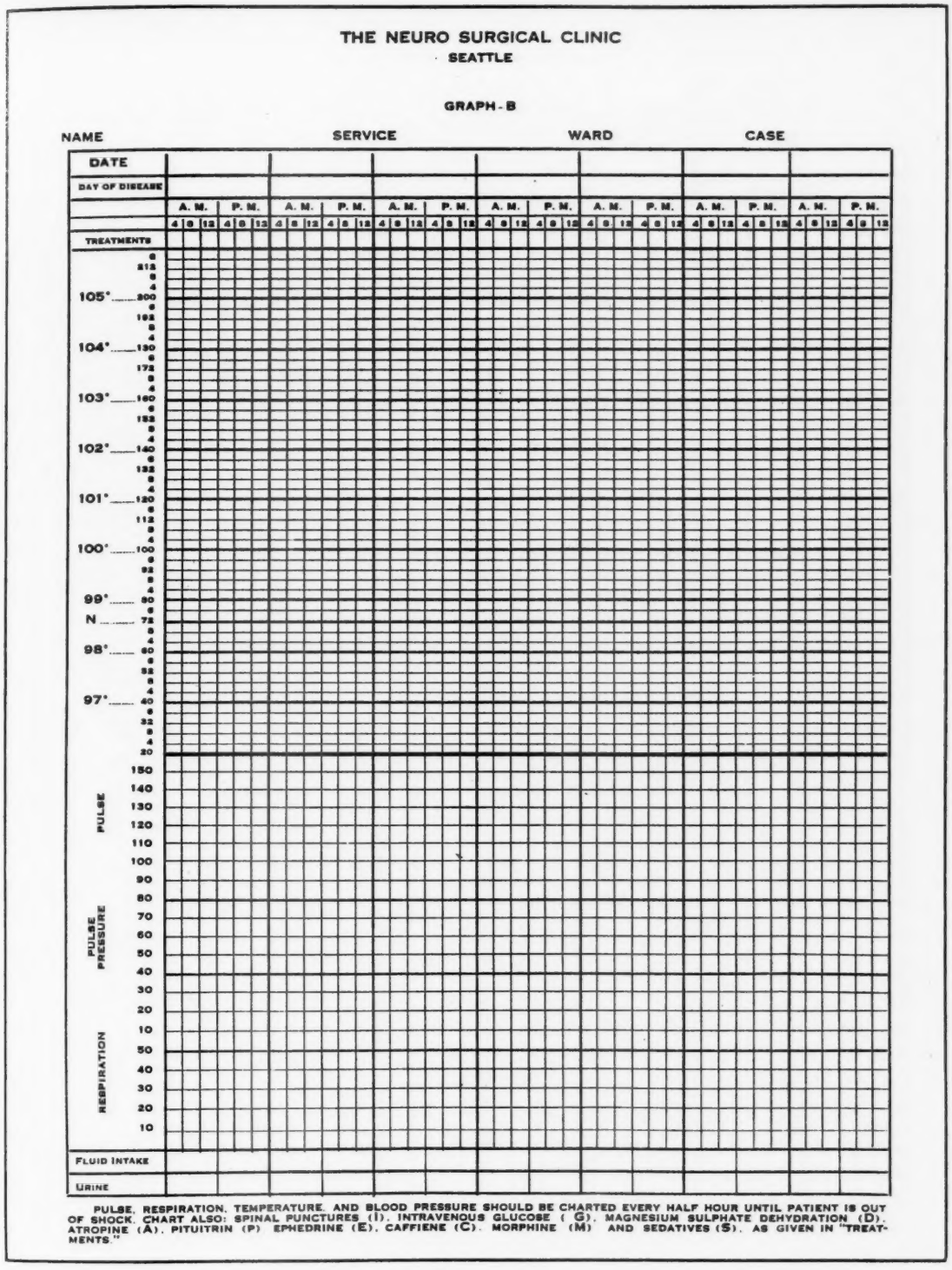

THE NEURO SURGICAL CLINIC
SEATTLE
GRAPH - B
NAME
SERVICE
WARD
CASE
DATE
DAY OF DISEASE
A. M.
P. M.
TREATMENTS
105°
104°
103°
102°
101°
100°
99°
N
98°
97°
PULSE
PULSE PRESSURE
RESPIRATION
FLUID INTAKE
URINE
PULSE, RESPIRATION, TEMPERATURE, AND BLOOD PRESSURE SHOULD BE CHARTED EVERY HALF HOUR UNTIL PATIENT IS OUT OF SHOCK. CHART ALSO: SPINAL PUNCTURES (I), INTRAVENOUS GLUCOSE (G), MAGNESIUM SULPHATE DEHYDRATION (D), ATROPINE (A), PITUITRIN (P) EPHEDRINE (E), CAFFIENE (C), MORPHINE (M) AND SEDATIVES (S), AS GIVEN IN "TREATMENTS."

Chart II.

in the patient who does not appear to be seriously injured. The patient may be conscious and have practically no complaints, but the insidious increase in pulse pressure and decrease in pulse without other marked symptoms, if allowed to go undetected, may frequently prove fatal. Such symptoms as these may easily be detected by the faithful use of Charts I and II.

POST-TRAUMATIC PROBLEMS

Treatment. One of the complications of a severe cerebrocranial injury is the so-called post-traumatic neurosis. To forestall this the surgeon should be alert for its early symptoms. Patients begin to brood over their injury, possibly realizing they do not function properly either mentally or physically. Soon suggestions, offered from the outside, so substantiate this idea that what was at first merely a notion or thought, gradually becomes a real belief. It is but a short step from this to the typical neurosis, that is, loss of independence and self-assertion, inability to assume former status in society, and a complete failure in readjustment to present environment. More functional symptoms, headache, generalized body weakness, sweating, fainting, loss of appetite and its subsequent loss of body weight, follow. Unless the course is checked, these patients become hopeless invalids and eventually are a total economic loss.

To prevent these results the surgeon should use every means at his command. The first and probably most important factor involved is the environment of the patient. The patient should have contacts only with those who are cheerful, intelligent, and strong physically. The subject of the injury should not be discussed until the patient has recovered from all of his actual symptoms. The injured parts should not be considered as seriously harmed, and as soon as the mental condition of the patient is such as to retain any facts, assurance of rapid recovery should be given.

The greatest problem is furnished by the family and friends. An early explanation of conditions and the effect of discussion upon the patient's future will usually break down any opposition and enlist their support. In cases where coöperation is not obtainable, the patient must be protected by the only other remedy, sedatives.

The second environmental factor is one of medicolegal character, responsibility for the accident, compensation, and possible criminal responsibility. Although this problem is the more difficult, since each case presents its own points and must be treated as such, generally, one need not worry much over the ultimate result. After the court has had its say, symptoms usually disappear.

Psychosis. Psychoses are traumatic, not post-traumatic. When the patient does not awaken to normal mental reactions and responsibilities but develops a psychotic pattern, the picture is entirely different from that which develops in post-traumatic neuroses. Traumatized brain tissue, especially in severe concussion, with or without bloody spinal fluid, is very likely to cause such psychoses. Many develop delusions, hallucinations, maniacal outbursts, severe vertigo, and unstable physical responses. Care, different from that of the neuroses, is necessary. Restraint, prolonged use of sedatives, and sanitorium treatment, not for days, but for months, are frequently necessary. Changes of personality (sometimes permanent), homicidal and suicidal tendencies must be detected and measures taken to prevent serious outbursts.

SUMMARY

In the conservative treatment of head injuries we have found need for certain immediate measures for the control of shock, hemorrhage, edema, and brain damage. These measures may be continued for a short time or in cases of prolonged unconsciousness must be continued for a longer period. Shock is the first and most important factor to control. After shock symptoms have subsided and hemorrhage and edema are under control one must make a careful survey to ascertain what

damage has been done to (1) the brain, meninges and spinal cord, and (2) other organs and viscera. This survey requires complete physical, neurologic, and ophthalmologic examinations, including the use of roentgenograms and spinal puncture. The results of these examinations plus observation of changes in symptoms will indicate the proper procedure.

Later treatment includes the prevention of post-traumatic neurosis. This depends upon the constant supervision of all personal contacts the patient may have during his convalescent period. Neurosis should never be allowed to develop. Psychoses associated with cerebrocranial injuries are the direct result of brain damage. They cannot be prevented; their presence is revealed as soon as the conscious period begins to appear. Treatment requires special and prolonged care and restraint.

While it is embarrassing at autopsy to find that a case should have been operated on, it is more embarrassing to find that an operation was unjustified. Experience has convinced us that about 90 per cent of the cerebrocranial injuries may be safely treated under these conservative rules.

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2. Swift, George W. Cerebral injuries due to external trauma. *Surg., Gynec. & Obst.*, 62: 340–346 (Feb. 15) 1936.
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As a general rule, it is held advisable in this clinic [Massachusetts General Hospital] to remove foreign materials, such as plates, nails, screws, and steel bands as soon as their purpose is definitely fulfilled.

CRANIOCEREBRAL TRAUMA

PRACTICAL CONSIDERATIONS FOR THE GENERAL SURGEON

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THE ever increasing number of head injuries, with their high mortality, disabling sequelae, and medicolegal complications, places a burden of great responsibility upon the general surgeon, as in many communities the service of a neurosurgeon is not available.

During the past twenty years, the principles underlying the treatment of brain trauma have changed considerably, the pendulum swinging from the extreme radicalism of immediate decompression in all serious cases to an opposite extreme of conservatism where operation is rarely done. The conservative treatment of head injuries, particularly by osmotic therapy, has undoubtedly greatly reduced mortality. Osmotic therapy is based primarily upon Weed's experimental work which demonstrated the value of intravenous hypertonic solutions in reducing intracranial pressure. Where formerly a routine decompression was done, now a routine system of dehydration prevails as a standardized treatment of brain trauma in many institutions. Standardization may have a place in certain routine medical procedures, but efficiency in therapy is apt to be embarrassed by standardization, and will continue to be so until we have a standardized pathology. The treatment of cerebral trauma cannot be standardized; the complexity and degree of traumatic lesions of the nervous system produce reactions which challenge all the art and resources of the experienced neurosurgeon in their interpretation and treatment.

INTRACRANIAL PRESSURE

The treatment of craniocerebral trauma resolves itself largely into the control of intracranial pressure. To evaluate clinical signs in determining the degree of pressure and its underlying pathologic cause, the surgeon must have at least some conception of cerebral hydrodynamics.

The Monro-Kellie doctrine, formulated in the early nineteenth century, introduced certain principles of physics relative to the flow of fluids through the closed cranial box. Of the three elements contained in the cranial chambers, nervous tissue, blood, and cerebrospinal fluid, an increase in the volume of one can be compensated only by a reduction in the volume of the others. If there be an increase of cerebral volume due to brain tumor, there is a compensatory reduction of cerebrospinal fluid and blood volume. The rigid cranial box is incapable of either expansion or contraction to compensate changes in the volume of the enclosed elements.

The cerebrospinal fluid is very active in this space adjustment mechanism, as it may be readily expressed from the cranial chambers into the general circulation to accommodate an increased volume of brain or blood. An obstruction occurring in the cerebrospinal fluid circulating system, such as the blocking of a ventricular outlet, may produce a high degree of intracranial pressure, depending upon the completeness of the block. In cerebrospinal fluid obstructions, the most active element in the pressure adjusting mechanism is impaired. The elaboration and absorption of cerebrospinal fluid normally is a nicely balanced mechanism which is influenced greatly by blood volume, osmotic, and intracranial pressure. An increase in cerebrospinal fluid osmotic pressure, caused by blood proteins, will increase cerebrospinal fluid volume. The

presence of blood in the cerebrospinal fluid, a common incident in head injuries, increases intracranial pressure by: (a) increasing cerebrospinal volume by raising its osmotic pressure; (b) mechanically obstructing the cerebrospinal fluid circulating channels; (c) volume of the extravasated blood.

TRAUMATIC CEREBRAL PATHOLOGY

Skull Fractures. In a consideration of traumatic cerebral pathology, skull fractures assume importance not because of the fractures per se, but because of their relation to an intracranial lesion. I have seen a number of extensive skull fractures with no immediate or late evidence of cerebral injury. From a medicolegal angle, however, the *x*-ray demonstration of a skull fracture often carries more conviction of serious injury than a neurologic report of impaired cerebral function. From a therapeutic or prognostic standpoint, the question of fracture is obviously subservient to that of brain injury, though a compound fracture assumes great importance in that it may be a possible source of intracranial infection. Fractures involving the accessory nasal sinuses or the ear, in which there is a leak of cerebrospinal fluid, must be considered serious, even in the absence of cerebral symptoms, because of the danger of infection.

Considerable concern often attends a depressed fracture, but, unless severe, a simple depression is of little significance in the absence of intracranial damage. It is often considered as an indication for immediate operation, and the surgeon who fails to operate on a depressed fracture has been considered negligent. In the absence of symptoms indicating intracranial involvement, the milder grades of depressed fractures require surgical intervention solely because of cosmetic effects.

The patient with head trauma, neurologically negative but with *x*-ray evidence of skull fracture, should receive hospitalization for a period of a week or ten days as a traumatic cerebral suspect, even though active treatment for such may not be necessary. In linear fractures of the temporoparietal region, one should remember the possibility of a developing hematoma with its delayed compression syndrome. The *x*-ray examination of a fractured skull should be conducted only when the patient may be safely transported to the *x*-ray room and stand the required manipulations.

Cerebral Trauma. Anatomically, injuries of the brain may occur in any region and to any degree. Concussion represents our most minor clinical conception of traumatic cerebral disturbance, consisting in a primary loss of consciousness immediately following a head injury. In concussion there may be very little cerebral disturbance with rapid recovery, or an associated brain laceration and hemorrhage may prolong the period of unconsciousness. Lacerations of the brain seldom occur without some degree of hemorrhage, and, since these lacerations more commonly occur in the cortex, blood frequently enters the cerebrospinal fluid pathways. A blockage of the cerebrospinal fluid circulating system always complicates the picture of cerebral trauma by increasing intracranial pressure. It is sometimes very difficult to differentiate between the pressure of a hematoma and the disturbance caused by an increase of cerebrospinal fluid.

Therapeutically there is little that we can do for cerebral laceration, but hemorrhage and its pressure effects demand specific treatment. Hemorrhage arising from lacerations of the cortex may tear the piarachnoid and allow blood and fluid to escape into the subdural space. Such a condition is probably much more common than we suspect, and perhaps a brief consideration of the subdural space might be pertinent in the question of such fluid accumulations.

The subdural space is a potential rather than an actual space between the arachnoid and the dura. Normally it is filled with a thin film of fluid which exerts an adhesive or cohesive action between the arachnoid and dura, and it is this cohesion between arachnoid and dura

which maintains the brain in its postural stability within the cranial cavity. An excess of fluid, blood, or air in the subdural space releases the arachnoidodural approximation, and the brain, deprived of this support, gravitates to the dependent portion of the skull. The action of such accumulations in the subdural space is not unlike that of similar ones within the pleural cavity. Accumulations of fluid or blood in the subdural space permitting a postural displacement of the brain throw traction upon the arachnoidodural attachments and upon the cortical veins which pass through the subdural space to enter the sagittal sinus.

A tear in the arachnoid following a cerebral trauma will frequently allow considerable fluid to enter the subdural space and cause an increase of intracranial pressure. As a rule such accumulations of fluid are not relieved by spinal puncture. The defect or tear in the arachnoid may eventually heal and the subdural fluid be gradually absorbed, or, if it contains blood, its volume may be increased by the osmotic action of the blood proteins. Occasionally a tear in the arachnoid does not close, but remains as a chronic arachnoidal fistula through which cerebrospinal fluid leaks into the subdural space, and there develops a postural instability of the brain, which becomes an important factor in the development of post-traumatic epilepsy, headaches, and personality changes. An arachnoidal fistula may be demonstrated by encephalography: air injected into the subarachnoid space escapes into the subdural space where it may be roentgenologically demonstrated. Sometimes air so injected may appear in the subdural space only after several days, but its appearance is always indicative of an arachnoidal fistula.

Subdural Hematoma. Two types of subdural hematoma are recognized following cerebral trauma. The acute type in which there is a rather diffuse extravasation of blood throughout the subdural space, and a chronic type which develops slowly, weeks or months after a head trauma. In the chronic type, the hematoma is usually well encapsulated, so much so that it often has the appearance of originating between the layers of the dura. Its slow development will usually suggest a neoplasm because of the gradual onset of pressure signs.

Extradural Hemorrhage. Fractures, particularly those involving the squamous portion of the temporal bone, may tear the middle meningeal artery as it passes along the meningeal grooves, with the development of an extradural hematoma. Extradural hematoma, gradually developing, produces delayed pressure signs. Thus a patient with an injury to the head with a concussion history may receive treatment and be discharged apparently in good condition, without signs of intracranial involvement, only to become unconscious hours later from the development of an extradural hematoma. This is why early head injuries with a history of concussion but no evidence of immediate pressure should be kept under observation for twenty-four or forty-eight hours, and for a considerably longer period if a fracture is demonstrated.

Cerebral Edema. The literature of craniocerebral traumas has placed much stress upon the question of cerebral edema, and increases of intracranial pressure following head injuries are often considered as being caused largely by a post-traumatic edema. Histologic examinations of the brain following trauma usually fail to reveal such an edema, the increased pressure being due to hemorrhage, accumulations of cerebrospinal fluid, or great increase in blood volume. When decompression was the standard treatment of head traumas, there would often be found, on opening the dura for the relief of pressure, a great collection of fluid which was then called "wet brain," and which was usually a collection of subdural fluid. At other times, instead of this free fluid, there would be a marked herniation of the brain, classified as "cerebral edema," but probably due to cerebral hemorrhage or congestion (increased blood volume, which was often intensified by straining during

the stage of excitement in the induction of an inhalation anesthesia). The advent of conservatism and osmotic therapy in the treatment of head injuries has greatly reduced the mortality in just such cases.

CLINICAL CONSIDERATIONS

In the attempt to correlate symptoms with an underlying traumatic cerebral pathology, we must consider the concussion, contusion, and compression of the older textbooks. Inasmuch as most actual brain injuries present an initial period of unconsciousness (concussion), it is wise to consider any accident to the head in which there is a history of a temporary disturbance of consciousness as a possible brain injury. Concussion is considered mainly as a "molecular disturbance" due to cerebral jarring, as there may be no pathologic evidence of organic disturbance. Contusion, however, may range from small petechial hemorrhages to severe lacerations with massive hemorrhage, presenting clinical signs of both local brain injury and general cerebral compression. It is often difficult to differentiate clinically between focal symptoms due to brain laceration and those produced by compression, and, since it is cerebral compression which most concerns the surgeon in the question of therapy, it is well to consider the clinical evidence of cerebral compression. Most medical men are familiar with the symptom triad of headache, vomiting, and choked disc, which characterizes most chronic elevations of intracranial pressure, but there is an entirely different clinical picture in acute pressure reactions.

Acute Elevations of Intracranial Pressure. Acute pressure elevations present a progression of symptoms which serve, with considerable reliability, in the determination of the degree of functional disturbance caused by an increased intracranial pressure. Many surgeons, however, rely almost entirely upon spinal puncture with manometric readings to determine cerebrospinal fluid pressure. Both are of importance. These symptoms of acute increases of intracranial pressure are largely caused by anoxemia, the increased pressure interfering with cerebral circulation. The effect of acute or rapidly induced pressure upon medullary centers is observed in characteristic reactions of pulse, respiration, blood pressure, and temperature. In slowly developing chronic elevations of intracranial pressure, circulation maintains a certain degree of adjustment; in these cases acute medullary reactions are not observed. With an acute head injury, after the abatement of shock, disturbance of medullary circulation due to pressure presents two phases with characteristic clinical features. The first phase represents a period of more or less successful efforts on the part of the organism toward circulatory compensation through vagal stimulation, with a slow pulse and an increased blood pressure. When the circulatory adjustment mechanism falters, due to increasing pressure, symptoms of medullary failure appear. The earlier stage in which compensation is effectual may be termed the *irritative stage.* If intracranial pressure continues to rise, a limit of pressure compensation is reached, and there then appear the signs of medullary failure which we term the *paralytic stage.* We have a similar picture, irritative and paralytic, in cortical reactions, manifested principally in disturbances of consciousness.

Medullary reactions in the stage of irritation show a pulse full, bounding, and progressively slowed. As intracranial pressure increases and circulatory compensation begins to fail, the pulse loses its slow bounding quality (pressure pulse) and becomes more and more rapid as decompensation advances in the paralytic stage.

Blood pressure is gradually increased with the slow pulse, often becoming very high, but with failure of the medullary centers, pressure fluctuates and finally assumes the characteristic Traub-Hering waves.

Respirations are slowed at first, but during the stage of decompensation gradually

assume the characteristic Cheyne-Stokes' rhythm.

Temperature during the compensatory or irritative stage is not markedly elevated, but with beginning loss of compensation it gradually ascends and may terminate in hyperthermia.

Cortical reactions to acute increases of intracranial pressure are characterized during the irritative stage by restlessness and irritability which gradually progress to excessive irritability, delirium, and, as compensation falters, pass into semicoma and finally profound coma.

elevations of intracranial pressure through the stages of compensation and decompensation. Lacerations or localized hemorrhage, however, may modify to some extent these reactions in individual cases, and variations in the clinical picture suggest their presence. In most head injuries where there is an increase of intracranial pressure, the patient in his conscious stage complains of headaches which are often severe. The eyegrounds will show a progressive congestion of the retinal veins which become torturous. Papilledema is not present in the early stages of acute pres-

TABLE I

ACUTE INTRACRANIAL PRESSURE

	Irritative Stage			Paralytic Stage			
Cortical:	Restlessness	irritability	delirium	semicoma	coma	profound coma.	
Pulse:	Slowed	slow and pounding		very slow	rapid	very rapid.	
Respiration:	Increased	slowed	very slow	stertorous	Cheyne-Stokes'.		
Blood pressure:	Increased	elevated	high	unstable	Traube-Hering waves.		
Temperature:	Normal	slightly elevated	elevated	high	hyperthermia.		

Keeping in mind these reactions, particularly as presented in Table I it is possible to determine with considerable accuracy the state of medullary and cerebral compensation in acute increases of intracranial pressure. In the stage where medullary compensation is effective, we have a relatively slow pulse, a somewhat elevated blood pressure, normal temperature, some reduction in respiratory rate, and restlessness. This is perhaps the most favorable period for treatment, and intelligent treatment will frequently prevent more serious degrees of compression. In the latter part of the compensatory stage, the patient passes into a delirium or a semicoma, the pulse is very slow, the blood pressure is high, respirations are slowed, and the temperature somewhat elevated. As medullary failure develops, coma deepens, blood pressure remains high or shows periods of instability, pulse becomes more rapid, and respirations are stertorous. In the terminal stage, there is a rapid pulse, Traub-Hering waves of blood pressure, Cheyne-Stokes' respiration, and profound coma.

The sectional clinical pictures presented here are typical of the reactions of acute

sure; it rarely develops before several days or a week.

A patient on admission may be in any stage of compression, with clinical pressure signs masked by shock. In severe head injuries, concussion is often overlaid with pressure symptoms, and the patient continues unconscious as a result of cerebral lacerations and hemorrhage. In the unconscious patient, an early neurologic examination showing a paresis or hemiplegia is indicative of laceration, and such a condition does not of necessity indicate surgical intervention. A paralysis, however, not originally present but gradually developed usually indicates a localized hemorrhage and developing hematoma. If, after recovery from concussion, there develops a secondary paralysis, with irritability and medullary reactions, then surgery is very definitely indicated—this is the typical syndrome of middle meningeal hemorrhage (extradural hematoma).

A patient admitted to the hospital in shock, following head injury, will not show typical clinical pressure reactions. Immediately after the period of shock has passed, if spinal puncture is done on the uncon-

scious patient and there is found an increase of cerebrospinal fluid pressure, hemorrhage is indicated; this is probably of a massive type if a high cerebrospinal pressure occurs within a few hours following the injury. Early coma with signs of medullary failure and a low cerebrospinal fluid pressure, determined by manometric readings, indicates extensive cerebral laceration without massive hemorrhage. While we usually look upon concussion as a rather brief period of unconsciousness due to jarring of the brain, we do have long periods of cortical disturbances due to diffuse minor lacerations, which are characterized by more or less prolonged periods of unconsciousness, semicoma, and delirium, without marked increase of intracranial pressure.

Immediate spinal puncture following head injuries will often show no increase of intracranial pressure, unless there be an initial hemorrhage. A gradual elevation of pressure is usually caused by disturbance in cerebrospinal fluid circulation, hematoma, or subdural collections of blood and fluid. Fractional pressure readings during spinal puncture are of considerable value in differentiating between excessive cerebrospinal fluid accumulations and the conditions just mentioned. I prefer the spinal mercurial manometer, as it seems more reliable for routine hospital work than the water manometer. The normal spinal pressure (recumbent position) with a mercurial manometer is from 6 to 10 mm. of mercury.

When spinal puncture is done (the patient being perfectly relaxed with the mouth open) and the pressure is found to be, for instance, 30 mm. Hg., 5 c.c. of cerebrospinal fluid are withdrawn cautiously, drop by drop; should manometric readings show a rapid fall in pressure (below 10 mm.), one may conclude that the increased intracranial pressure is not due to an excessive amount of unobstructed cerebrospinal fluid. If, however, 5 c.c. are removed and the pressure is not materially decreased and the fluid flows freely, then we may conclude that the pressure is due to an increase of cerebrospinal fluid. Sufficient fluid is then withdrawn to reduce the pressure to normal, and spinal taps continued as long as the patient shows signs of irritability or an increased cerebrospinal fluid pressure.

Eye Findings. As previously stated, papilledema is not observed in an acute increase of intracranial pressure; it may, however, appear in the subacute or chronic stage. Early visual field defects are usually indicative of direct trauma to the optic tracts or nerves, though in later stages these changes may be due to cerebral compression. Engorgement of the retinal veins, with tortuosity, is commonly observed in the acute stages, but is of little value in determining degrees of pressure. The condition of the pupils, however, is of considerable importance. A dilated pupil on one side is often significant of a localized hematoma confined to that side, though occasionally this may be misleading. It is particularly significant, however, if both pupils be found originally active with one slightly smaller and later the smaller pupil be found dilated; a localized pressure (hematoma) on the side of the dilated pupil is apt to be disclosed at operation. Dilatation of both pupils, however, is usually indicative of a marked degree of generalized intracranial pressure, but this seldom develops before the patient is in coma.

TREATMENT OF CRANIOCEREBRAL TRAUMA

Most victims of severe head injuries are in shock when they reach the hospital, and treatment should be primarily directed to the relief of this condition. During the stage of shock, it is difficult to interpret signs of increased intracranial pressure, and the serious condition of the patient should not be jeopardized by extensive examinations nor the manipulation necessary for *x*-ray examination. In the control of shock, intravenous glucose serves to support the general circulation and controls, at least to some extent temporarily, an increase of intracranial pressure. Glucose is indicated particularly when there has been great loss

of fluids with reduced blood volume (as in the sweating of shock and hemorrhage). It should not be used to reduce intracranial pressure with increased blood volume unless used with diuretics or active purgation to remove excessive body fluids. Rest, warmth, and fluids are of major importance during shock.

When the systolic pressure reaches 80 mm. Hg. and the patient is dry and warm, fluids should be restricted. At this time, a careful neurologic examination should be made to determine the extent of the underlying pathology. Reference to the table of pressure symptoms will throw considerable light on the question of pressure, and marked deviation from this picture suggests reactions due to local brain injury. A spinal puncture should be done with great care (having the patient relaxed and perfectly quiet if one is to depend upon manometric pressure readings); if the pressure is high, cerebrospinal fluid may be drained slowly until approximately a normal pressure is obtained. In the withdrawal of fluid, a pressure reading should be made after the removal of each 5 c.c.

A low initial cerebrospinal fluid pressure immediately after the subsidence of shock is not uncommon. If the patient shows clinical signs of increasing pressure, a second spinal puncture should be done after six hours to determine whether there has developed an increased cerebrospinal fluid pressure.

Spinal punctures are of value in that they remove excessive cerebrospinal fluid, blood, and particularly blood proteins, which are often responsible for a high osmotic pressure and excessive fluid. It is in this condition, that of excessive cerebrospinal fluid, that treatment by spinal drainage is most effective. In cerebral injury, however, much of the pressure may be due to hemorrhage, and fractional cerebrospinal fluid pressure tests will serve to differentiate between the pressure of fluid and that of hemorrhage or cerebral congestion. Cerebral congestion due to increased blood volume is an important factor in intracranial pressure, and in this condition dehydration or osmotic therapy is of great value, as it definitely decreases blood volume. It serves also to reduce cerebrospinal fluid volume by raising the osmotic pressure of the blood.

It has been amply demonstrated that the administration of intravenous glucose is attended with a secondary rise in pressure. This secondary rise is not experienced after sucrose, although to obtain the same dehydrant effects practically twice the volume of sucrose should be administered. Sucrose has a marked diuretic action, and a secondary rise in intracranial pressure is prevented by the induced diuresis, which is usually abundant after intravenous sucrose. In the average case, about 300 c.c. of 50 per cent sucrose is given by vein and may be repeated as indicated, depending largely upon the variation in clinical symptoms and cerebrospinal fluid pressure as determined by manometric readings.

In any type of dehydration treatment, whether it be with glucose, sucrose, or by alimentary magnesium sulfate, it is important that the fluids removed should not be replaced rapidly by giving fluids too freely by mouth, as patients become very thirsty. Dehydration treatment should, however, be carefully controlled by blood volume determinations, as in many instances harmful effects have resulted from excessive dehydration. A reduction in blood pressure as well as cerebrospinal fluid pressure will usually attend successful dehydration. If there is not an amelioration of pressure symptoms following dehydration, the surgeon is justified in concluding that the increase in intracranial pressure is due to cerebral, subarachnoidal, subdural, or extradural hemorrhage, and the question of surgical intervention arises. The answering of this question often taxes the judgment of an experienced neurosurgeon. We may conclude that an increase of intracranial pressure, which is not being satisfactorily influenced by spinal drainage and intelligent dehydration offers little prospect of improvement without surgery. When sur-

gery is definitely indicated, as in various types of hematoma, it is often attended by brilliant results, but in many instances cerebral lacerations and hemorrhage exist to a degree that precludes recovery regardless of the type of treatment.

A certain number of craniocerebral injuries present at no time a demonstrable increase of intracranial pressure though there may be marked restlessness, delirium, or coma; such reactions are usually due to cerebral lacerations and subcortical hemorrhage of a subpressure degree. Some have bloody cerebrospinal fluid without increased pressure, and the clinical signs are not those typical of pressure. The absence of increased pressure at any one time does not preclude a subsequent rise, and a careful consideration of clinical symptoms is necessary from day to day; also an occasional spinal puncture to check pressure and remove bloody fluid is advisable. The treatment of non-pressure head traumas is largely supportive: fluids, complete rest, and restriction of noise and bright light as much as possible. Restlessness is best controlled with phenobarbital. Headache always suggests pressure, but, with pressure controlled, aspirin and codeine are often effective in its relief.

In compound fractures of the base, with discharges of cerebrospinal fluid from the ear or nose, avoid manipulation as much as possible; do not irrigate nose or ears. A few drops of 50 per cent alcohol, several times daily, in the external auditory canal, and a light cotton plug offer probably the safest treatment in cerebrospinal fluid otorrhea. Do not tampon the nose to control a discharge of cerebrospinal fluid. An abundant escape of cerebrospinal fluid from the ears or nose is a contraindication for spinal puncture. After such a discharge stops, spinal drainage may be indicated to control pressure. An increase in temperature, however, calls for an immediate puncture and cell count. Any suggestion of cerebrospinal fluid infection calls for immediate cerebrospinal drainage by frequent spinal punctures and large doses of sulfanilamide. As a prophylactic in fractures involving the ears or accessory sinuses, the use of sulfanilamide is recommended.

Operative Treatment. The surgical treatment of cerebral traumatic lesions is primarily directed toward relief of intracranial pressure, except possibly in the case of compound fractures in which early surgery is required in the prevention of infection. Little, if anything, can be done by surgical methods for cortical or subcortical hemorrhage or lacerations. Surgical treatment, however, is very definitely indicated in subdural hematoma and extradural hemorrhage. Occasionally a liberal subtemporal decompression will serve to relieve the pressure of a deep-seated hemorrhage, provided active bleeding has stopped.

Subdural hematoma of the acute type, in which there is an accumulation of blood and fluid in the subdural space, is often bilateral in location and a bilateral decompression is advisable. The side operated on first should be that corresponding to a unilateral dilated pupil, if such exists, or the side contralateral to a unilateral Babinski. If neurologic indications are inadequate to determine the side of approach, and there is *x*-ray evidence of a fracture in the lateral aspects of the skull, the decompression may be done over this area; otherwise a subtemporal exposure is advisable.

Under local, avertin, or oil-ether anesthesia (never under inhalation anesthesia) a burr opening is made through the bone and enlarged sufficiently for adequate inspection. The dura is opened, and if there be a subdural collection of fluid or blood, it is immediately evident. Extradural hematoma from middle meningeal hemorrhage will, of course, be evident before opening the dura, providing the exposure is made in the temporal region, where the middle meningeal vessels are more commonly injured. In each case, the clot is removed as far as possible and the bleeding point ligated, as is often required in extradural hemorrhage. In subdural hematoma, active

bleeding is seldom encountered unless surgically induced; evacuation of the clot is the essential objective. This can usually be accomplished through a fairly small decompressive opening by suction and irrigation, but, if this cannot be done without injury to the brain, a flap is advisable. The complete removal of small fragments of an extradural or subdural clot is not necessary, particularly when additional surgical trauma is unavoidable.

Recurrence of subdural hematoma is exceptional. If a small exposure on one side fails to show a definite pathologic condition, simply a bulging brain, do not attempt further exploration on that side if you are satisfied that a hematoma does not exist. Such a procedure, if carefully done under local anesthesia, is attended with little, if any, shock.

If no pathology is found on the one side to account for the intracranial pressure, the opposite side should be explored in a similar manner. If both sides are carefully explored with no evidence of a hematoma, and it is decided that decompression is indicated for the relief of pressure, a wide subtemporal decompression may be done on the right side of a right-handed individual.

The removal of an intracranial hematoma, epidural or subdural, may be the determining factor in recovery from a head injury, although it may not completely relieve all signs of intracranial pressure, and a postoperative continuation of dehydration and spinal drainage may be indicated. Restlessness, irritability, headaches, and nuchal rigidity, as well as increased cerebrospinal fluid pressure, should always be indications for continued treatment.

COMPOUND FRACTURE

The early surgical treatment of a compound fracture is imperative in the prevention of infective sequelae and should be done immediately after the abatement of shock. The most satisfactory treatment is complete débridement of the scalp laceration and the removal of bone fragments. If the dura is intact, it should not be opened unless it is blue, giving evidence of an underlying hematoma. Under such conditions it may be opened, but with extreme aseptic care. The clot should be removed as thoroughly as possible without inflicting further trauma, the dura closed, and the scalp sutured without drainage. In compound fractures of more than twenty-four hours' standing, drainage should be instituted and the dura not opened; if the dura is found to be lacerated, further trauma is to be avoided, as adhesions have probably sealed the subarachnoid space and should not be disturbed.

A hemiplegia or a paresis is not an indication for surgical intervention, providing the paralysis is immediate. If, however, the paralytic symptoms develop subsequent to the primary examination, they may indicate compression by hematoma, and so constitute indications for surgical exploration. However, if there is no progressive increase in intracranial pressure and no increase of the paralysis, surgery had best be deferred.

MORTALITY

In all severe types of head injuries, there will be a high mortality regardless of the nature of the therapeutic procedure used. The mortality statistics of head injuries, particularly as they are related to treatment, are often misleading because of difference in classification. A group of minor injuries under any type of treatment will give better statistical results than a like number of more serious injuries. In certain hospitals having a similar rate and type of admissions over a number of years, and using during this period a similar classification, the mortality figures indicate a considerable reduction with the employment of osmotic therapy and conservative treatment. In institutions in which a standardized treatment of head injuries has been adopted without regard to individual pathology, as is occasionally found in institutions in which head injuries are not under the supervision of a neurosurgical

staff, we may conclude that where the standardized treatment is osmotic therapy or spinal drainage alone, the mortality figures will not be as low as they might. There will probably be more post-traumatic sequelae than would occur if each patient were treated on an individual pathologic basis as evidenced by the clinical reactions of that particular trauma.

SEQUELAE OF BRAIN TRAUMA

Undoubtedly a large percentage of those surviving a severe brain injury have sequelae producing varying degrees of disability which may be more or less permanent. There is no question but that the desire for compensation serves to intensify functional symptoms, particularly anxiety states. A patient who has had employment interrupted because of head injury and is burdened with economic responsibilities is a fit subject for functional disturbance; he probably has been told that his brain was injured and expects the worst in sequelae (mental impairment or insanity). The problem of reëmployment, particularly with the fear of impaired ability, contributes strongly to the post-traumatic neurosis.

Though it is desirable in all head injuries to effect compensational adjustments and rehabilitation as early as possible, it must not be forgotten that there has been a definite brain injury and there may be a very real organic basis for complaints. Each patient suffering from a head injury should have a careful neurologic examination, probably several, at intervals of a month or more in which due attention is given to the separation of organic, functional, and psychotic disturbance. Subjective complaints should not be classified as neurotic until an organic basis has been definitely eliminated by expert examination.

Organic Sequelae. *A paralysis or paresis* which has not cleared up within three months following brain injury, and has developed spasticity with exaggerated and pathologic reflexes, will probably remain a permanent disability. A neurologic examination will determine whether a paralysis is organic or functional (hysterical). Whatever the basis of the hysterical paralysis may be, it will usually clear up under suggestion, compensational adjustments, and rehabilitation efforts.

Cranial Nerve Injuries. Traumatic head lesions more commonly affect the oculomotor, facial, and vestibulo-auditory nerves. Injuries to these nerves are usually in their peripheral portion, and the degree of injury may be determined readily by examination. The vestibular apparatus is commonly injured in fractures of the base, and the complaint of vertigo, or dizziness, should always call for vestibular function examination. If these tests are negative, dizziness, at least, may be due to other factors, such as subdural fluid accumulations, or a cranial defect with its characteristic instability syndrome.

Visual Defects. Disturbances of vision following head injuries are not uncommon. Visual field defects usually indicate a definite lesion of the visual pathways or optic nerves. A concentric constriction of the visual fields with other signs of increased intracranial pressure should immediately cause one to suspect a subdural hematoma. Tubular vision, however, should be considered on a hysterical basis. Engorgement of the retinal veins, an increased redness of the disc, or papilledema always suggests an increase of intracranial pressure and should receive neurosurgical investigation.

Post-Traumatic Headaches. Post-traumatic headaches are a common sequela of head injuries. They are usually due to an elevation of intracranial pressure caused most commonly by either an increase of cerebrospinal fluid pressure due to an obstruction in its channels of circulation or a collection of subdural fluid. Post-traumatic headaches are often relieved by repeated spinal punctures, though subdural accumulations of fluid, which are responsible for so many post-traumatic sequelae, usually require subdural drainage.

Post-Traumatic Epilepsy. When convulsive phenomena occur, there is a tendency in the profession to give a very pessimistic prognosis and place the blame on some pretraumatic neuropathy. This is entirely unjustified, because in post-traumatic epilepsy there exists a definite pathology which can usually be corrected. A common cause of post-traumatic epilepsy is an arachnoidal fistula through which fluid leaks into the subdural space producing a postural instability of the brain. With this postural instability the brain moves in response to gravity, throwing traction upon traumatic corticordural attachments which, in time, become hyperirritable as a result of these insults. Convulsive phenomena sooner or later appear as cortical stability becomes more and more impaired. It may require months or years for this epileptic mechanism to develop. A correction of the underlying physical factors will usually result in the recovery of cortical stability and cessation of epileptic phenomena.

Depressed Fractures. As previously stated, a depressed fracture of small magnitude is not an indication for immediate surgery, unless it is a compound fracture or there are signs of an underlying trauma. Depressed fractures, however, may require later surgery because of cosmetic reasons or an anxiety state. When such surgery is required, a fracture may be elevated or conformity restored by some plastic procedure when the bone must be sacrificed. The writer prefers the use of celluloid, which is perfectly tolerated, to bone or cartilage grafts which are often absorbed.

Chronic Subdural Hematoma. Chronic subdural hematoma is a more or less encapsulated hematoma within the subdural space. It may occur months after even a slight head injury. Subdural hematomas develop gradually, become encapsulated, but tend to expand, and are commonly bilateral. Symptomatically they are difficult to differentiate from cerebral neoplasm; there is usually a period of gradually increasing headaches, unexplainable attacks of vomiting, papilledema, and contraction of the visual fields.

Chronic subdural hematoma is characterized by signs of a chronic increase of intracranial pressure and usually by an absence of localizing neurologic findings. In the late stages, these patients often develop periodic disturbances of consciousness which at times may be rather profound. There is practically always an increase of cerebrospinal fluid pressure, and differential pressure readings will show a pressure which, from a high point of 30 or 40 mm. Hg., will be reduced to zero upon removing a few c.c. of fluid. It is extremely dangerous, however, to remove cerebrospinal fluid in any quantity when this is attended by a rapid fall in pressure.

With encephalography, a unilateral subdural hematoma will show the ventricles collapsed and displaced to the contralateral side. With subdural air and lipiodol insufflation it is possible to outline a subdural hematoma. In ventriculography or subdural air insufflation, a subdural hematoma is often recognized by the appearance of blood clots when the dura is nicked.

The surgery of chronic subdural hematoma had best be left to the experienced neurosurgeon. Patients with chronic increases of intracranial pressure following head injuries present problems in diagnosis and treatment which, as a rule, are not willingly accepted by the general surgeon, and inasmuch as these lesions are not acute they may be safely transferred to a neurosurgeon.

Functional Sequelae. Hysteria is perhaps one of the most frequent functional disturbances following craniocerebral trauma. An hysterical paralysis can usually be differentiated from the organic type by its flaccid nature, lack of spasticity, and by sensory changes which do not conform to a typical organic distribution and which may vary from day to day. Obviously, their treatment is not surgical. Many vague functional disturbances may be the subject

of complaint during the process of compensation adjustments; it is usually possible, however, by careful examination, to differentiate between organic and functional disturbance, and in functional disturbances early compensation adjustment is desirable.

Post-Traumatic Psychic States. Serious psychic disturbances are not common following brain trauma, and we have no reason to believe that the development of a schizophrenic disturbance following a trauma is necessarily due to that trauma, any more than a brain tumor can be etiologically connected with trauma. Observations in either case do not seem to support even a remote traumatic relation. Character changes, however, do follow head trauma and this is probably the most common type of psychic disturbance. There is much evidence to support the assumption that personality and character changes following brain trauma have an organic basis. They may be directly due to brain injury or to a subacute elevation of intracranial pressure which has never been properly relieved. I have seen, in a number of instances, profound disturbances of this kind result from head injuries, and I have also seen spectacular relief follow the drainage of large quantities of subdural fluid.

It seems necessary to emphasize the importance of completely relieving any increase of intracranial pressure in head trauma cases before a patient is discharged from the hospital. The persistence of some degree of irritability, nuchal rigidity, and headache, no matter how well the patient may appear otherwise, should be considered as a contraindication to discharge until such a time as cerebrospinal fluid pressure has become normal.

SUMMARY

1. Intracranial fluid dynamics in relation to acute elevations of intracranial pressure are briefly presented for the benefit of the general surgeon who is compelled to treat craniocerebral traumas.

2. A chart of acute intracranial pressure reactions which served well, in the writer's experience, in the evaluation of clinical signs in relation to cerebrospinal fluid pressure is incorporated.

3. Traumatic cerebral pathology is discussed in relation to clinical signs, and indications for specific types of treatment: cerebrospinal fluid drainage, dehydration, and surgical intervention.

4. Post-traumatic sequelae are considered in relation to their underlying pathology, their treatment, and their prevention.

FRACTURES OF THE LOWER JAW

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FRACTURES of the mandible occur most frequently because of trauma; those due to tumors and infections are rarely seen. Traumatic injuries resulting in fractures of the lower jaw are usually produced in fist fights, automobile accidents and miscellaneous falls. Less than 5 per cent of mandibular fractures result from accidents which occur while the individual is engaged in a gainful occupation. Fewer than 100 cases have been reported in the last twenty years to the Maryland State Accident Commission. When such injuries do occur in the course of employment, it is in such people as jockeys, due to falls from horses; steel workers, struck by air hammers; seamen and stevedores, injured by various lifting devices; and boxers, hit by a fist.

The most frequent sites of fracture, in the order named, are: through the junction of the body and the ramus, the bicuspid region, the molar region, and the symphysis. A fracture of the condyloid process is usually associated with a fracture of the body of the mandible on the opposite side. Fractures of the coronoid process are rarely seen. Unilateral fractures occur twice as often as the bilateral type. Combinations of fracture sites in bilateral fractures are in the following order: bicuspid region on one side and at the junction of the body and ramus on the other; the body of the mandible on one side and neck of the condyle on the other; and through the body of the mandible on each side.

The diagnosis of these fractures is not usually difficult. There is a history of traumatic injury, with pain, swelling, improper occlusion of the teeth and a partial loss of function of the lower jaw. Clinical examination reveals abnormal mobility and crepitus. Radiographic examination clearly discloses the lines of fracture. In the radiographic examination, lateral jaw plates are taken of both sides. If the fracture occurs through the condyle, anteroposterior views are necessary to show any lateral displacement, while if the fracture is at the symphysis, an occlusal view is taken with an intraoral film.

The treatment of fractures of the mandible varies according to the site of fracture, and the presence or absence of teeth. In all cases, simplicity of treatment is stressed. Intraoral impressions, complicated splints, undue manipulation of the parts, and cumbersome head apparatus are avoided. The fracture should be reduced as soon as possible following the injury. If reduction is completed before muscular contraction takes place, the operation is considerably simplified. When muscular contraction and swelling has taken place, complete reduction cannot be accomplished immediately; a gradual reduction must be resorted to.

In fractures where teeth are present, the simple intermaxillary wire eyelet method, using the coarse grade of angles brass ligature wire, is used. A small loop is made in the wire and the ends are passed through the embrasure formed by the bicuspid teeth. One end is then brought around the lingual side of the first bicuspid, and the other around the lingual side of the second bicuspid. Both ends of the wire are brought completely around the bicuspids and twisted tightly together. This gives a wire loop firmly attached to the two bicuspid teeth. This same procedure is followed on the opposite side of the jaw and also in the maxilla. Pieces of wire are then passed through the eyelets which are attached to the maxillary and mandibular teeth on each side. This brings the teeth into occlusion, and makes the maxillary

teeth a splint for the reduction of the lower jaw. The intermaxillary wires have a tendency to stretch, and must therefore be tightened every two or three days. The wires are kept in place for five or six weeks.

By this method of reduction, proper occlusion of the teeth is insured, no time is lost in making splints, and no uncomfortable head bandage is necessary. Some patients with fracture of the mandible have many teeth missing, so that it is necessary to use other combinations of teeth for the wires. It is occasionally necessary to place the eyelet wire on a single tooth instead of using two teeth; this can be accomplished by passing the wire around the tooth twice, and then twisting the ends together. Should it be desired to apply traction anteriorly or posteriorly, the eyelet wires can be so placed as to make this possible. If upper and lower teeth are missing, which allows an upward displacement of a posterior fragment, a piece of dental impression compound is slightly

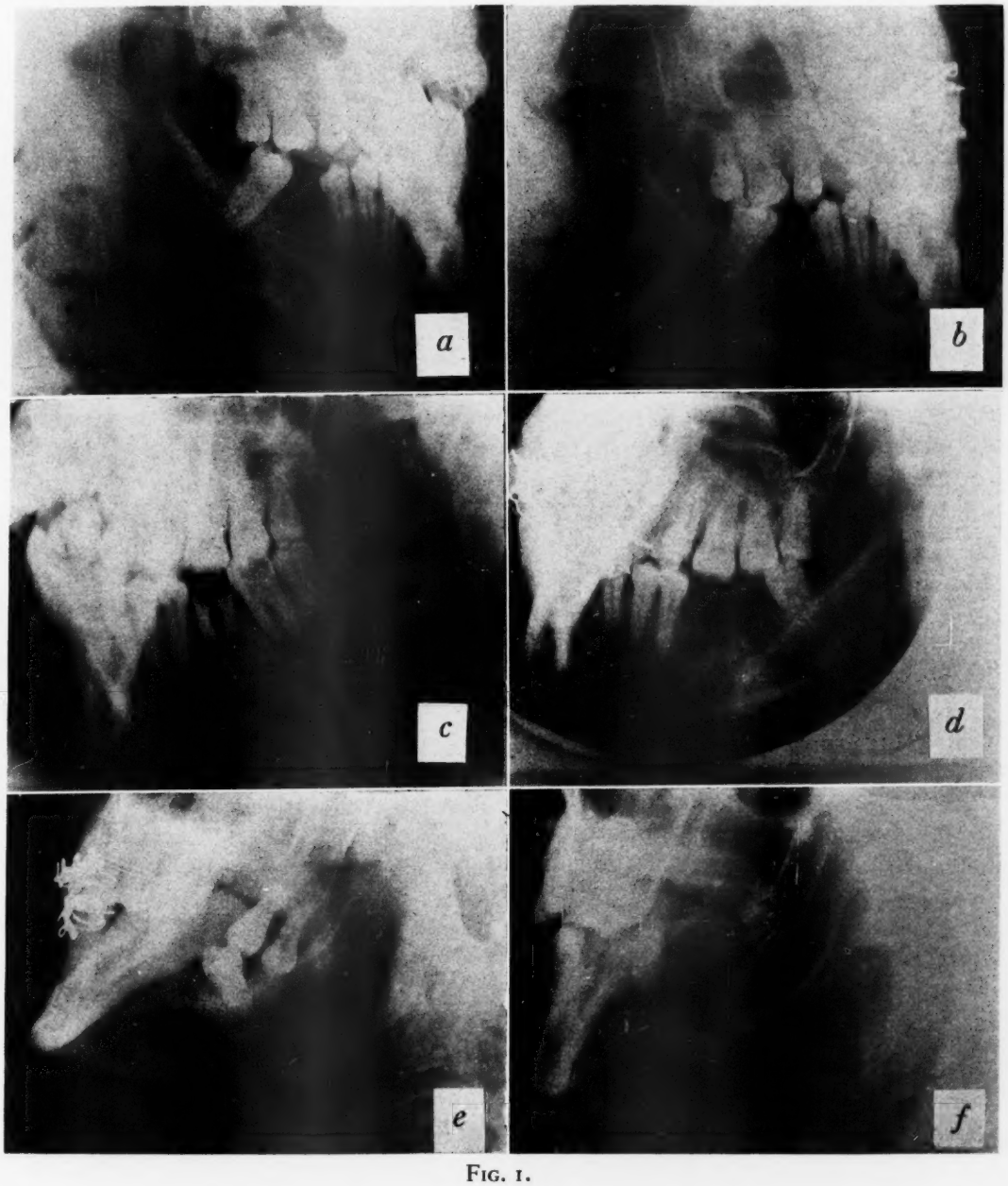

Fig. 1.

softened and placed over the fragment. The mouth is then closed and the fragment is manipulated into its proper relationship. The compound is moulded to hold in position during fixation.

If upper teeth are missing where an eyelet should be placed, a piece of stainless steel wire is twisted around the two teeth nearest one another, making a bridge of double twisted wire over the space. The intermaxillary communicating wire is then placed over the bridging wire and through the eyelet on the lower teeth. This will satisfactorily immobilize the part if it is further supported by other wires as near the area as possible.

In cases where all upper teeth are missing and an artificial upper denture is present, the lower teeth are wired to the upper denture and then supported by a Barton bandage and two inch strips of adhesive tape. The upper denture holds the lower teeth in proper occlusion and the bandage gives the necessary additional support.

Where there is a fracture of the neck of the condyle, a piece of impression compound or gutta percha, about 2 mm. thick, is placed between the teeth on that side and the jaw immobilized by intermaxillary wires. The condyle will always assume its proper position as soon as muscular relaxation takes place. No trouble has been experienced with ankylosis, either partial or complete. It is striking to note how few complications and bad results follow fractures of the mandible. There have been no cases of non-union and seldom bad anatomic or functional results.

In fractures of edentulous mandibles, reduction and fixation are accomplished by the open reduction method. Under local anesthesia, the mucous membrane on the buccal side is incised so as to reflect sufficient tissue to expose well the line of fracture. The lingual mucous membrane is then reflected away from the mandible. With a dental drill, holes are bored through the bone, and either silver or stainless steel wire is threaded through the openings. The wire is then twisted to hold the parts together, and the mucous membrane is sutured together with black silk. This wire is not removed. In most cases it is not necessary to use any form of splint or head bandage for additional support.

The oral cavity should be properly cleansed before any fracture is reduced. All remaining roots and excessive salivary calculus should be removed. A tooth in the direct line of fracture should be removed unless it is the last remaining tooth and is needed to retain the posterior fragment in proper position. If the tooth is retained at the time of reduction, it should be removed later, as soon as the healing is sufficient to maintain the posterior fragment in position.

Practically all of these fractures are reduced under local anesthesia, conduction injections of 2 per cent novocaine being given wherever possible. If considerable manipulation is necessary, as in cases of delayed treatment, intravenous evipal is the anesthetic of choice, since it is of short duration and leaves no nausea.

All fractures of the mandible where teeth are present, are compounded through the alveolus of the tooth sockets; however, osteomyelitis is rare. If incision is indicated, it is best done extraorally in order to establish better drainage and allow the mucous membrane within the mouth to heal. The application of heat by means of short wave diathermy has been of some advantage.

The patient must be properly nourished during the period of treatment while the jaws are immobilized, but naturally, this must be done by means of liquids.

The following is a sample menu of a liquid diet for patients with immovable jaws. It contains approximately 2,410 calories, protein 80 Gm., fat 130 Gm., and carbohydrate 230 Gm.*

8 A.M. Grapefruit juice, 200 c.c., with 1 tablespoon glucose (karo).
Eggnog made with 200 c.c. milk, one egg, one teaspoon sugar, vanilla flavoring.

* The menu outline was prepared by the Diet School of the University Hospital, Baltimore, Maryland.

Cereal gruel made with one-half cup oatmeal or other cooked cereal, one-half cup milk, one-half cup cream, one teaspoon sugar, or more if desired.

10 A.M. Cocoa made with 200 c.c. milk, one teaspoon cocoa, two teaspoons sugar.

12 M. Strained vegetable soup, 200 c.c.

Iced chocolate made with 200 c.c. milk, one-half square chocolate, two teaspoons sugar.

Orange juice, 200 c.c., with one tablespoon glucose (karo).

2 P.M. Eggnog, as above.

6 P.M. Cream soup, 200 c.c., made with one-half cup vegetable purée, one-half cup milk, one-half cup cream, one square butter, thinned with water if necessary.

Pineapple juice or other fruit juice, 200 c.c.

Cocoa, as above; malted milk may be used occasionally instead of cocoa and chocolate milk.

8 P.M. Eggnog, as above.

If additional nourishment is needed, cream may be used with the milk in the preparation of milk drinks. Tea and coffee have no food value unless cream and sugar are added. The clear karo used in the fruit juice may be dissolved in hot water.

It is routine to add 10 gr. of calcium gluconate and 10 drops of drisdol to the daily ration throughout the six weeks of treatment.

Most of these cases are ambulatory, and after the second or third week are able to go back to their former occupations, if their work is not too strenuous.

SUMMARY OF TREATMENT

1. Early diagnosis and treatment.
2. Simplicity of treatment with complete reduction and fixation.
3. Complete radiographic examination to locate all fractures.
4. Serologic test for lues and urinalysis for diabetes.
5. Maintain proper mouth hygiene.
6. Sufficient and proper nourishment.
7. Proper elimination.

THE PREVENTION AND CORRECTION OF FACIAL DISFIGUREMENT

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THE first aid given to a facial injury may determine success or failure in the final result and too often the opportunities present at the time are overlooked. In bony injuries there is frequently a lack of appreciation of this and a resulting lack of correction of the underlying deformity. Bony displacements should be corrected immediately or within the first ten days. In the upper jaw they must be done within this time or they can never be corrected. If the face were not exposed and if the personality of the individual were not largely expressed in the motions of the face, less subjective distress would result from the deformity. In the correction of injuries of an extremity, function is a major consideration, but in facial injuries appearance is of prime importance.

The two major groups of injuries of the face are those of the soft parts and those of the underlying structure. They may occur separately or combined with one another.

Where the condition of the tissue permits, primary suture within the first few hours after injury is the method of election for tears through the full thickness of the eyelids, ears, lips, alae and trap-door flaps, and this may simplify secondary restoration when needed. Early repair in most other incised wounds may be helpful in shortening the healing time but primary suture does not always do away with the necessity of secondary repair. Such a repair will give as good a restoration on a wound that is healed by granulation. Too often a secondary repair can easily make the original scar unnoticeable, while the scars resulting from the first suturing remain glaringly visible throughout life. (Fig. 1.)

Except in the repair of eyelids débridement should be confined to the removal of the devitalized tissue only. While the danger of large skin sutures should be rigidly avoided, the bringing and holding of incised tissues in apposition or approximation over gauze packing is always in order. In relatively clean wounds this may be done with subcuticular sutures which may be of fine silk, engaging only the derma, or of chromic catgut, including deep masses of tissue. Where packing is used, or where the tissues are easily but not necessarily accurately approximated, strapping with adhesive plaster is good surgery and may prove sufficient. Scratches and abrasions, especially those containing cinders or oil, should be cleansed by scrubbing with a stiff brush. Under novocaine infiltration this cleansing is rendered more efficient.

Foreign bodies (Fig. 2) should be removed from any lacerated wound when possible. Digital examination of every large wound or probing of the smaller ones must be done in a search for the foreign body. When necessary, the wound may have to be enlarged for proper examination.

In dressing these repaired wounds, a soft, damp sea-sponge will give equalized distribution of bandage pressure when properly applied.

In fractures of the face, the teeth are the most accurate means for securing stationary fixation of mobile fragments of

tooth bearing bone, and such fractures are easily and accurately fixed in place by fastening the teeth of one jaw with the corresponding teeth of the other. The only other accurate means is by direct bone fixation, passing prepared tendon or fine silver wire through drill holes in the bone.

With but few exceptions, fractures of the lower jaw can be held in position by simple wiring of the lower teeth to the upper in proper occlusion. An uncontrolled posterior fragment without a usable tooth can be immobilized by one of the following methods: Direct bone wiring, fixing the coronoid process by a screw through the zygoma, some sort of dental splint, or a silver wire engaging the posterior border of the ramus, protruding through the skin and giving backward traction. Absence of teeth in important positions in the upper jaw can be met by fixing the fragments to a wire arch about the teeth or in the lower by circumferential wiring of the fragments. In a true symphysis fracture there is a tendency for the fragments to rock laterally in spite of interdental wiring. This can be remedied by a low-bone wiring of the two fragments through an incision just beneath the chin, or by a Gilmer posterior band splint. Fractures of the condyle, neck, or intramuscular part of the ramus are best treated conservatively by interdental wiring only. A backward displaced chin fragment in a double fracture can be held forward with an extension bar attached to an upper dental splint, or a plaster head cap, or it can be held by forward traction over a pulley. A Gunning type open-bite splint is suitable for fractures in which there is downward displacement due to pull of the mandibular-hyoid group of muscles. In an edentulous jaw, a lower dental plate is a suitable splint with the fragments held to it by circumferential wires. The lower plate should then be wired to the upper or to an open-bite splint for fixation.

Fig. 1. This scar can be greatly improved by secondary operation. However, the transverse scars left by sutures at primary repair cannot be corrected. Such a primary repair actually makes the permanent deformity worse.

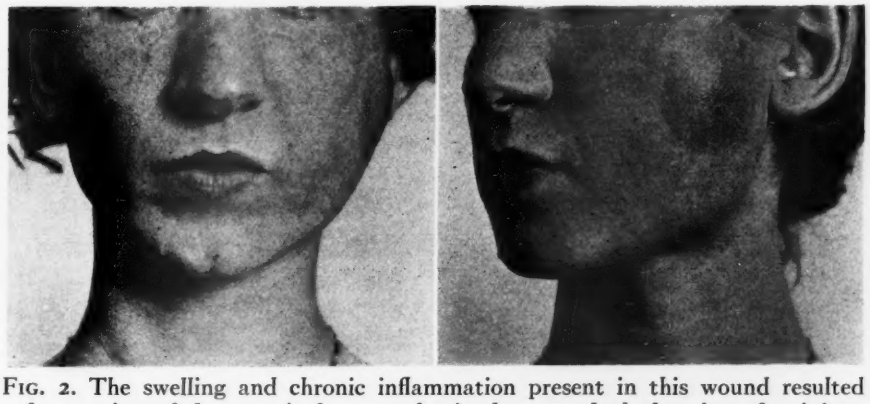
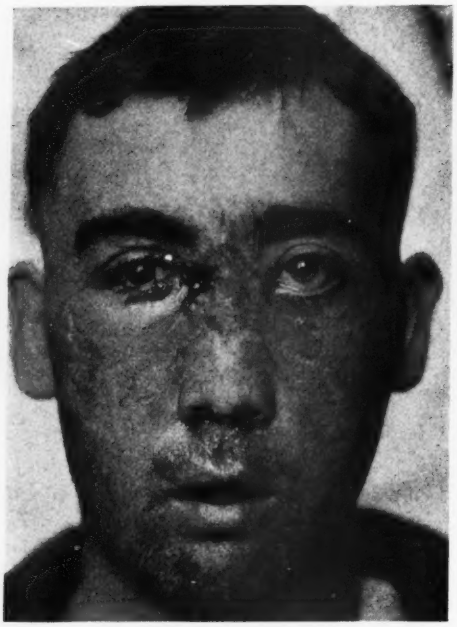


Fig. 2. The swelling and chronic inflammation present in this wound resulted from a piece of glass, one inch square, having been overlooked at time of an injury eight years previously.

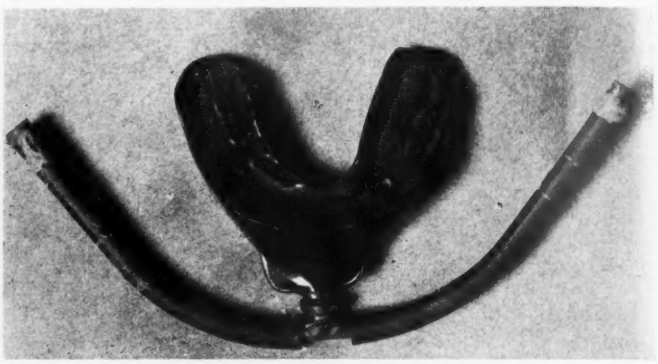
Fractures of the maxilla differ from fractures of the mandible and most other bones of the body because the displacement is due to the trauma itself and to gravity rather than to muscle pull. The only exception is the zygomatic process which is pulled downward by the masseter muscle. The term maxilla, as used here, includes the lacrimals, the lateral masses of the ethmoid, the vomer and parts of the sphenoid, as well as the maxillae, nasal and malar bones and the zygomatic process. This is necessary because fractures of this region follow no definite anatomic lines, but usually traverse more than one of these bones.

Fracture of the alveolar process is best treated by replacement of the fragments and fixation with interdental wiring or dental appliances. There is seldom complete separation of the process because of the bridge of closely adherent mucoperiosteum. In these fractures there may be displacement of the outer or inner table, there may be displacement of the whole mass of the alveolar process and attached teeth, or a fracture of the body may traverse the alveolar process.

Stellate fracture of the palate with an upper tilt requires reduction by pressure on the floor of the nose only.



A



B

Fig. 3. A transverse facial fracture showing the abnormal elongation of the face due to separation of the fragments of suture line. The treatment of such a fracture involves the maintaining of occlusion by interdental fixation to insure the proper relationship of the jaws. In addition to this, the approximation of the fragments must be obtained. Occasionally the tension of the masseter muscle is adequate. An elastic support passing around the head and under the chin is often sufficient. Occasionally apposition of the fragments must be obtained before interdental fixation is accomplished. This may be done with an inverted Kingsley splint and elastic upward pull for forty-eighty hours.

Other fractures of the maxilla may be divided into three or four distinct general types, although such a loculate mass of bone can be broken in many directions.

There is first, the rare vertical type with separation of the two halves of the maxilla. A second type is the lower transverse fracture, which partially or completely passes through the antra with or without radiating lines through the palate or the alveolar process. Usually little tearing of the oral mucosa occurs. There may be separation and free mobility of the palate or a portion of it. Impactions should be broken up, especially when the bone is driven upward and backward, for there may result impairment of hearing due to crowding of the Eustachian cushions and orifices. This is far more important than the persistent malocclusion and facial deformity.

The upper transverse fracture (Fig. 3) passes through the orbits, nasal cavity, pterygoid and zygomatic processes, with little tearing of the soft tissue covering.

There is usually abnormal mobility of the dental arch with a sagging and elongation of the face, which, if untreated, becomes progressively worse. Extension through the skull and dura, injuries to the sixth nerve, globe or optic nerve, may complicate this fracture and must continually be kept in mind.

The principles of elevation of the fragments and reëstablishing normal occlusion are common to both these transverse fractures. The teeth or a denture splint should be wired in place to fix the dental arches, except perhaps in those rare cases where there is no displacement and little excursion. But in the latter, all chewing must be prohibited and the patient must be carefully watched for an increase in false movement during at least a three-week period. The immediate fixation of the teeth in occlusion may embarrass respiration as a result of edema of the nasal mucosa, palate and pharynx. However, the wires may be attached to the teeth at the time of reduction, while fixation and occlusion are done two to three days later.

Usually, little attention need be paid to the contents of the orbit. The downward displacement of the globe is corrected when the bony wall of the orbit is elevated into normal position. In the lower transverse fractures, the intra-oral mass should be moulded into position and care taken to insure patency of the nasal passages.

In gravity displacement of transverse fractures, a Kingsley splint with a slight elastic upward pull will correct the sagging, and dental fixation can later be established. A period of two to three weeks is the maximum time for upward traction, as there is danger of overcorrection or inaccurate correction of the comminution, causing a permanent malocclusion. Interdental wiring should, therefore, be done before the end of the third week.

Fractures of the malar bone are a fourth common type. They are usually impacted with displacements inward and backward or slightly downward, outward and backward. There may be disturbance in opening the mouth due to interference with forward motion of the coronoid process. Associated with these displacements is a disturbance in the continuity of the lower or lower and outer border of the orbit, and also of the orbital floor. A downward and backward displacement of the globe results and may easily be overlooked.

The impaction can be freed and the bony mass replaced through the antrum. The mucosa is incised above the three molar teeth and if the bony wall is intact, a chisel is driven upward into the antrum. Accurate replacement may be accomplished by instrumental leverage within the antrum combined with digital manipulation without. The cavity is packed firmly and thoroughly with iodoform gauze, lightly impregnated with balsam of Peru, so placed as to maintain the bones in position and also as to be easily withdrawn two or three weeks later. A Dakin's tube may be included in the packing for daily irrigation.

Many of these fracture displacements of the malar bone can be replaced by inserting an elevator beneath the temporal fascia, into the zygomatic fossa and elevating the body and temporal process of the bone outward. This method is simple to execute, but there may persist a flattening of the malar prominence or a slump or retraction of the globe. More perfect correction might be obtained in some cases by a combined intramaxillary and temporal approach.

A blow against the upper bridge of the nose tends to drive the nasal bones either backward between the frontal processes and under the frontal bone or even the cribiform plate of the ethmoid or it may flatten the nasal roof and push out the frontal processes with the anterior lacrimal crest and perhaps the lacrimal bones. The orbit is thus impinged upon and the distance between the inner canthi is increased. At the same time the intact nasal septum is crumpled and should be straightened at the time the bridge of the nose is raised. Any torn or detached nasal cartilage should be repaired with intranasal or extranasal suture.

A B C

FIG. 4. A, with this type of injury, where there is loss of tissue and ragged contused lacerations, often very little can be accomplished at primary treatment other than the removal of all oil and dirt that might produce tattooing. This should be removed by scrubbing under anesthesia if necessary. B, the result six months later, following spontaneous healing, no repair having been made. C, following secondary repair, which consisted of replacement of scar epithelium on forehead with a free full thickness skin graft and an excision of the rough scars with careful suture.

The bridge of the nose as well as the backward displacements of the frontal process of the maxilla can be pried forward with a urethral sound inserted into the nasal cavity. Likewise a crushed frontal sinus wall can be elevated by inserting a chisel into the sinus just beneath the orbital border. The nasal bridge may retain its contour after the bones are raised and the septum straightened, but often an intranasal bar attached to a dental splint may be required. The flattening and lateral spread with separation of the inner canthi can be corrected by wire mattress sutures through the full thickness of the nasal wall and septum, traversing the fracture lines and tied over a lead plate on each side of the nose. If there is a persistent tendency to deviation to one side and the nasal bones and septum are not shattered, a silver wire can be made to engage the lower border of the bridge and traverse the opposite nostril to be attached to a bicuspid or first molar tooth. When too much comminution is present, a splint attached to a denture is more effective in fixing the nose. The anterior nasal spine can be freed if displaced or impacted by driving a chisel through the upper fornix.

Except as a support for the septum, intranasal packing is of little help in holding displaced bone fragments in exact position.

Facial injuries to the nose in childhood may develop into marked deformities during the growing period. It is a good plan to make forcible forward pressure on the bony bridge from within on a theory that a later hardening and distorted growth of the septal cartilage may be induced by the abnormal back pressure of imperceptibly depressed bone.

The keynote in the care of face fractures is exact reduction and retention before solidification has taken place. No attached fragments of bone should be removed. The result should not be compromised by a strained attempt to close the soft tissues, particularly in gunshot wounds.

Late corrections of bony depressions can be compensated for by cartilage transplants and resurfacing by split thickness grafts, full thickness grafts, or where necessary, by hair-bearing grafts. Losses from the body or ramus of the mandible can be filled by bone grafts. Losses from the alveolar portion of the upper jaw can be compensated for by dental prostheses. Before applying the latter it may be necessary to free the soft tissues and reline the sulcus with a split skin graft.

SUMMARY

To recapitulate, all fractures should be reduced and the fragments held in their normal position before solidification commences. For those of the lower jaw and the tooth-bearing part of the upper jaw the reduction should be as early as possible. Compound fractures of the lower jaw should be drained by external incision.

With most injuries of the soft tissue it is better practice to let the wound heal spontaneously than to indulge in early débridement, or run the risk of suture scars. Tattooed wounds, whether from road oil or gunpowder, should have the foreign matter removed early; the former by vigorous scrubbing with a stiff brush after anesthetizing the surface, the latter by cutting out each individual grain with a needle-pointed knife. It might be quite difficult to remove all coloring matter from incised wounds such as come from the edge of a stovepipe, but this can be excised and repaired later. In seeking gross foreign bodies in an open wound, the finger will sometimes detect what *x*-ray and vision will miss.

ABDOMINAL CATASTROPHES RESULTING FROM EXTERNAL VIOLENCE*

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ABDOMINAL injuries include a great variety of possibilities, some of which may be easily overlooked by the examining physician when dealing with a patient who has lately suffered some physical mishap.

It is a simple matter to diagnose a penetrating or perforating wound of the abdomen when the wound or wounds are evident on the body of the patient and are somewhere in the abdominal parietes. It must not be overlooked, however, that the wound of entrance in injuries of the abdomen may be in the chest wall, in the back, in the pelvis or upper thigh. This is especially apt to be true in wounds of the chest at or about the level of the diaphragm. This problem is made more difficult by the anatomic arrangement and behavior of the diaphragm, which is fixed to the walls of the torso at its periphery, but changes its position constantly as far as its central portions are concerned. This is of prime import on the left side, because the spleen and stomach are just under the diaphragm and rise and fall with it. It is evident, therefore, that if one could know whether the diaphragm were elevated or depressed at the moment the wound was received, it would be helpful in determining whether or not the abdominal cavity had been entered.

In a crushing injury without external wounds some abdominal structure may be penetrated by fragments of bone, either from the lower ribs or the pelvis, or the diaphragm may be lacerated by fractured rib ends.

If there is a perforating wound the course of the missile is apt to approximate a straight line between the wound of entrance and exit, but not necessarily so. A spent bullet or soft bullet fired from a cheap gun may be diverted by obstructions that it meets. If there is no wound of exit, roentgenograms are invaluable provided the wound is a gunshot one. One would expect the course of the missile to be in the neighborhood of a line between the wound of entrance and its position as designated by the film.

In penetrating wounds caused by stabbing roentgenograms are of little help. The length of the knife, the force with which it was driven and its direction are important. These points are often difficult to evaluate, however. Quite often, in a stab wound of the abdominal wall, a piece of omentum sticking through the skin will be ample proof that the peritoneal cavity has been entered. Even when there is proof that the abdominal cavity has been penetrated, it is still possible that no intra-abdominal structure has been injured, but there are only two ways to determine this; exploration and waiting, and it is folly to wait if the wound is a recent one.

There is another group of possible abdominal injuries in which the diagnosis may be much more difficult, the so-called subcutaneous injuries. Here there is no open wound in the walls of the torso, but as the result of compression due to crushing injuries or concussion due to a fall or a sharp blow, there may be laceration or severe contusion of some abdominal structure.

It is important to bear in mind a number of anatomic facts. These have to

do with the relationship between the intraperitoneal contents of the abdomen and the extraperitoneal ones. In considering the abdominal wall as distinct from the abdominal cavity one must remember the relation of the abdomen to the chest, the back and the pelvis. There are many structures behind the peritoneum and any of these may be injured. In this category the kidneys are most important and are more often injured than any other postperitoneal structures.

In injuries of the upper abdomen the pancreas may be harmed. If such an injury is not recognized, repaired, or amply drained, a serious postperitoneal infection may result. The same thing is true of injuries to the large intestine, especially where the mesentery is short or absent and where penetration or postoperative leakage is causing postperitoneal infection.

Wounds of the urinary bladder and of the rectum may be either intraperitoneal or extraperitoneal or both. If extraperitoneal wounds of the bladder or rectum are overlooked and not amply drained, very serious and rapid infection takes place.

There are many other structures in the postperitoneal region, but most of them escape injury. If the aorta or vena cava or any of the large vessels of the aorta are entered, death usually takes place rapidly. Occasionally, however, following concussion or contusion of the abdomen a large postperitoneal hematoma may produce very confusing and disturbing symptoms. Sometimes such a hematoma may extend for some distance between the folds of the mesentery.

Abdominal injuries are usually classified in their relationship to the peritoneal cavity, bearing in mind the abdominal wall, the diaphragm and chest, the pelvis and its contents and the retroperitoneal region. Because we have been thinking chiefly of the abdominal structures that are covered in whole or in part by the peritoneum, we lay great stress on the factor of penetration or perforation of the peritoneal cavity. That is a rational attitude, because infection or bleeding in the peritoneal cavity is always serious and must be safeguarded against. It is very important, however, to keep in mind the abdominal wall, the pelvis, the diaphragm and the different structures that are found behind the peritoneum. The abdominal cavity and the peritoneal space are not quite the same. Abdominal injuries, therefore, may be classified as follows:

1. Open wounds that do not penetrate the peritoneum.
   - A. Wounds of the abdominal wall.
   - B. Injury of retroperitoneal structures.
   - C. Wounds of extraperitoneal pelvic structures.
2. Penetrating or perforating wounds.
3. Injury of any abdominal structure without penetration or perforation of the skin.

No classification is entirely satisfactory or all inclusive. The peritoneal cavity may be penetrated through the rectum or from the cavity of the uterus or from the bladder by instrumentation, or the stomach wall may be penetrated by instrumentation through the esophagus. Such penetrating wounds are not common, but their possibility should not be ignored.

Following injuries of the abdomen, hemorrhage and infection are the two great dangers. Both may be absent, one or the other may be present or the two may be found in the same patient. Hemorrhage is usually primary, it may be recurrent, but it is rarely secondary except as a late manifestation of infection.

Severe bleeding does not take place in all serious abdominal injuries. If a large mesenteric vessel is severed the loss of blood may be rapid and alarming and as it escapes into the peritoneum there is little tendency to clotting. Hemorrhage from the liver or spleen may be severe. Most of the blood entering the liver is under a low pressure and penetrating wounds may not bleed profusely. It is somewhat different with the spleen. The pressure in the

splenic artery is about that in the aorta and any considerable rent in this organ is apt to bleed dangerously.

The most unusual severe hemorrhage I have seen was due to severance of the hepatic artery in the right edge of the hepatoduodenal ligament. This was caused by a truck wheel passing across the upper abdomen of a child. The artery was severed just as it passed from left to right ventral to the right edge of the body of one of the vertebrae.

Some years ago, a patient came into the University Hospital of Baltimore, several hours after receiving a stab wound in the lower abdomen to the right of the midline. He was moribund and died almost immediately after admission. Examination showed a penetrating wound of the abdominal cavity without any intra-abdominal damage, except that the peritoneal cavity was filled with blood. The knife had severed the inferior epigastric artery and the entire hemorrhage had been through the abdominal wall rent into the peritoneum.

If there is an incised wound of the greater or lesser curvature of the stomach, hemorrhage may be severe. More often the stomach is penetrated or perforated away from its borders and the amount of bleeding is surprisingly small. Bleeding from the omentum is apt to be arrested by some clotting in the neighborhood of the injury.

If there is severe laceration of either kidney there is considerable bleeding. If there is no free escape of the blood either into abdomen, pleura or outside, the bleeding will usually be arrested short of an immediate fatality by pressure of the surrounding structures.

An individual bent on self destruction will occasionally, while standing, attempt to shoot himself through the heart with a revolver in the right hand. He may locate the heart by the apex beat and shoot too low, the bullet passing through lung, diaphragm, stomach, spleen or kidney, depending upon the course of the bullet. Such patients do not die immediately and present a difficult operative problem.

In any combined thoracic and abdominal injury blood may escape from the chest into the abdomen through a rent in the diaphragm. Considerable time may be lost at operation looking for the point of bleeding in the abdomen, if this fact is not kept in mind.

Postperitoneal hemorrhage may be excessive, moderate or slight. The aorta may be injured and if the opening is small the pressure of surrounding tissues may limit the hemorrhage. If such an opening is found at operation it may be possible to compress the aorta by manual pressure and suture the wall, turning the edges of the wound out and placing interrupted silk sutures close together. Ligation of the abdominal aorta is a rather futile procedure. If the vena cava is injured it is much easier to control bleeding, because the pressure is low. A lateral ligature or suturing may close the rent in the vessel wall. It may be necessary to ligate it. Pressure should be applied temporarily, while the next step is being considered. The portal vein is rarely injured.

Hemorrhage from mesenteric vessels is always serious, because of the loss of blood and because severance of a large mesenteric vessel usually calls for resection of the gut supplied by the injured vessel. Any very considerable collateral circulation cannot be expected in dealing with the blood supply of the intestine.

Hematomas in the mesentery and behind the peritoneum present a very serious problem. If no very important vessel is severed it is best to let these areas alone. If, on the other hand, active bleeding is going on it should be explored. This is especially difficult to determine in penetrating wounds of the upper abdomen with postperitoneal hemorrhage in the neighborhood of the pancreas. If blood is escaping slowly from the pancreas, it is, perhaps, better surgery to drain the pancreatic region after suturing any perforation in either wall of the stomach. It must be remembered that the splenic vessels run from right to left, very close to the upper

margins of the body and tail of the pancreas.

In traumatic surgery of the abdomen, hemorrhage and shock are the causes of early death. Death after several days is usually due to infection. Peritonitis may be only a part of the picture. Postperitoneal infection is, perhaps, the graver condition, because it has little tendency to become localized and is difficult to approach and drain after the infection is well established. Postperitoneal infection is more likely to be encountered in definite regions and because of definite lesions. Infection in the neighborhood of the pancreas is especially to be feared.

Another location where postperitoneal infection is to be prevented, if possible, has to do with the colon. If a perforation in the extraperitoneal portion of the large intestine is overlooked or if found and sutured and not adequately drained, serious or fatal infection may occur. After separating the lateral gutter in searching for and repairing a perforation in the colon, this region should be drained by ample stab drainage lateral to the colon. This maneuver may confine infection to this region without gross contamination of the peritoneum and places the trauma and drains away from coils of small intestine. This area may be peritonealized, in part, by suturing the lateral gutter of the colon ventral to the drains.

If the kidney has been lacerated and perinephritic infection is evident, it is essential to provide drainage. One must be prepared to deal with the kidney if such drainage has to be done as hemorrhage from a lacerated kidney may be primary, recurrent or secondary.

Extraperitoneal infection may be seen in the neighborhood of the bladder and rectum. These will be considered under these subjects.

Peritonitis as a complication of abdominal injuries depends upon a number of factors. If a hollow viscus is opened peritonitis is imminent. These structures are the biliary duct apparatus, pancreas and duct, stomach and intestines and the intraperitoneal portion of the urinary bladder. Infection may be carried in from the outside. If a considerable amount of bile escapes along with blood from a laceration of the liver, peritonitis may occur.

In any perforation of hollow viscera the time of operation is often the determining factor as to recovery or death. The chance of recovery depends on many things, but every hour that passes between receipt of the injury and operation is precious time lost.

In our experience in Evacuation Hospital No. 8 in France during the World War, this time factor could be relied on with considerable exactness. If a patient came in with the signs and symptoms of late, diffuse peritonitis and with a penetrating wound of the abdomen and if twelve to eighteen hours had elapsed since the injury, operation had little if any effect on mortality. If, on the other hand, twelve to eighteen hours had elasped since receipt of the abdominal wound and if diffuse peritonitis was not present, these patients did much better if treated expectantly. There were exceptions to both rules, but the number of hours that had elapsed was often the determining factor as to operative or expectant treatment.

The importance of the time factor is considerably altered by the level of the wound in the gut; wounds in stomach, duodenum and upper jejunum give a better outlook than those in the lower jejunum, ileum and colon. The lower and wider the openings and the greater the number of them, the worse the prognosis, other things being equal.

When a patient is seen with an evident or possible abdominal injury the problem may be a simple, complicated or difficult one. If the injury is a recent one and there is a wound of the abdominal wall, it must be determined as soon as possible whether the peritoneal cavity has been entered, whether there are other injuries and whether the patient is in shock, and

if so whether the shock is due to hemorrhage, trauma or is psychic.

After a preliminary inspection and brief history, it is important to determine the nature of the pulse and the blood pressure. Unless there has been active hemorrhage or the patient is in shock, a careful examination should be made. One must determine whether the wound in the abdominal wall is a penetrating one or not. In the absence of perforation and where there is no protrusion through the wound, and when the patient is not shocked, the abdominal wound should be explored sufficiently to determine its depth and direction. A probe is a dangerous and misleading instrument in penetrating wounds of the abdomen. In gunshot, stab and gutter wounds the abdominal wall around the injury should be carefully cleansed and with infiltration anesthesia the wound excised and opened widely enough to inspect it thoroughly. If it does not enter the peritoneum treat it as a simple wound. If it enters the peritoneal cavity cover the wound with a sterile dressing and take the necessary steps for an exploratory laparotomy. If the patient has a gutter wound it should be treated in the same manner, with this provision in mind. Gunshot and stab wounds do not usually cause enough trauma to bring about rupture of intra-abdominal structures by concussion or compression. A gutter wound, while it may not penetrate the whole thickness of the abdominal wall, may have been caused by trauma sufficiently severe to produce laceration of intra-abdominal structures by concussion or compression.

If the patient presents the signs and symptoms of perforation when seen, as little time as possible should be lost in bringing him to the operating table. Active concealed hemorrhage may be evident. Perforation of the abdomen may be present. If there is a penetrating gunshot wound the position of the missile may be determined by palpating it under the skin, but usually an *x*-ray will be necessary. There may be protrusion of gut or omentum through the wound. If sufficient time has elapsed since receipt of the injury, there may be evidences of peritoneal infection, but the important thing to bear in mind is the fact that in penetration of the abdominal cavity, seen early, concealed hemorrhage is not often evident and there are few symptoms of peritoneal infection. This is the most favorable time to operate for intra-abdominal injury, whether for perforation of a hollow viscus or hemorrhage, or both. There is, therefore, a grave obligation to determine at once whether or not the abdominal cavity has been penetrated. If it has, there is no choice of procedure. The mandate is clear-cut and imperative. The abdominal cavity should be explored and at once. If the patient is in shock, immediate steps should be taken in preparation for blood transfusion. It may be possible to do an auto transfusion as soon as the abdomen is opened, but this cannot be relied on. The peritoneal blood may be contaminated.

If an inspiration anesthetic is to be given it is important to remember that vomiting of stomach contents may lead to fatal aspiration pneumonitis. Spinal anesthesia is in many instances an excellent solution of the anesthetic problem, but shock is a contraindication. A good anesthetist is often more important than the choice of anesthesia.

If there is a penetrating wound through the back, seen early, and if it can be determined that there is no perforation of the peritoneum, watchful waiting is, in most instances, the safer procedure.

When there is a penetrating wound of the pelvis the problem is always a serious one. It is difficult to diagnose perforation of the peritoneal cavity at the bottom of the peritoneal pelvic pouch in either sex. It is important to remember that the cul de sac between the vagina and rectum descends lower than the peritoneal pouch between the bladder and rectum. Even if the peritoneum is not injured, extraperitoneal perforation of either rectum or

bladder calls for immediate operation. Here drainage is more urgently needed than repair.

If a number of hours have passed since injury there may be no doubt of perforation, as infection or bleeding or both may be determined by the patient's condition without recourse to exploration of the wound or other aids in diagnosis, such as catheterization of the bladder and *x*-ray. Vomiting, fast pulse, abdominal rigidity, pain, tenderness and shock present a clinical picture that is unmistakable. It is a pity that this evidence is ever necessary. It is most important that no physician be responsible for the loss of time that allows these symptoms to develop.

If there is a penetrating wound anywhere in the neighborhood of the abdomen the examining physician is on guard, but in the so-called subcutaneous injuries of the abdomen much time may be lost by patient and physician, unless both are alert to the possibility of mishap.

Any sharp blow on the abdomen, a fall or any crushing injury applied to the torso may open a hollow viscus, may lacerate a solid organ or may tear across mesentery or omentum. The liver, because it is large and near the surface of the body, is often injured. The spleen is well protected by its position, but because of its friability is often lacerated. The kidneys may be contused or lacerated; a full bladder, either urinary or biliary, may be ruptured. Any portion of the stomach or intestine may be torn and large rents in the mesentery or omentum are encountered. Laceration of liver or spleen is the most common injury, so that in the majority of cases hemorrhage into the peritoneal cavity is the outstanding cause of the signs and symptoms that may be evident. Hemorrhage may be slow, moderate or rapid. There may be some discoloration of the abdominal wall at the place where the maximum force was applied. On the other hand, the abdominal wall may be without any evidence of trauma. Localization of tenderness and pain may be enough to be helpful in diagnosis.

If any considerable amount of blood is in the peritoneal cavity many clinicians believe that the abdomen presents a "doughy" feeling on palpation. There may be dulness in the flanks also. The blood shows little tendency to clot, except in the immediate vicinity of the laceration. There may be some blueness about the umbilicus. If the abdomen is full of fat all of these signs may be difficult to elicit.

The symptoms are often more clear-cut if bleeding is active. The evidences of concealed hemorrhage are easy to elicit—shock, paleness, weakness, low blood pressure and a rapid pulse. Determining the hemoglobin is not apt to be helpful in recent hemorrhage, because the blood has not had time to increase its bulk by absorption of fluids.

If a hollow structure is torn, there is apt to be little bleeding and the signs and symptoms are those of infection, either in the peritoneal cavity or behind it or below it in the pelvis. If any considerable time is lost the outlook for these patients is bad and such injuries give a very high mortality, especially if diffuse peritonitis is present.

In addition to hemorrhage and infection there is another type of mishap that may follow this type of injury. The diaphragm may be lacerated and intra-abdominal structures may be protruded into the pleura. Just what may happen depends upon which side of the diaphragm is torn, and the size of the rent. On the right side a torn diaphragm and lacerated liver may produce hemothorax and may be very misleading. On the left side a portion of the stomach, spleen, colon or coils of small intestine may be extruded into the pleural cavity. In addition any of these structures may be torn. This latter complication is rare, however. The outstanding symptoms are those of obstruction to the structures extruded into the chest due to constriction caused by the margins of the hole in the

diaphragm. Occasionally the rent will be large enough not to cause obstruction and diagnosis may be overlooked.

Still another kind of abdominal injury due to compression is caused by fractured rib ends that may perforate or lacerate diaphragm and intra-abdominal organs or produce injury to either rectum or urinary bladder following fracture of the pelvis.

Wounds of the Urinary Bladder. The bladder is both an extra- and an intraperitoneal viscus. In small children, who have a shallow pelvis, it occupies a relatively high position. It rises up in the abdomen just to the extent that it is full of urine. In low abdominal injuries it is apt to be penetrated. Rarely, if full, it will be ruptured. It is most apt to be injured by penetrating injuries from without or by spicules of bone. The opening may be intra- or extraperitoneal, or both. Intraperitoneal injuries produce a rapid and fatal peritonitis. Extraperitoneal injuries are followed by urinary infiltration, destructive cellulitis and widespread necrosis, unless drainage is promptly done.

Blood in the urine or no urine on catheterization is an important finding when the penetrating wound is in the neighborhood of the bladder. Following a crushing injury of the pelvis the posterior urethra may be torn across without injury to the bladder and extraperitoneal urinary infiltration may cause widespread damage. Following a blow or fall, blood in the urine may result from kidney damage with a normal bladder. If there has been considerable bleeding into the bladder, from whatever source, the blood may clot and interfere seriously with keeping the bladder clean.

If there is an intraperitoneal perforation of the bladder, the hole should be closed by a double row of sutures and a retention catheter left in the bladder. Whether the peritoneum needs to be drained will depend upon the amount of peritonitis found and the presence of other injuries.

In extraperitoneal injuries of the bladder, suprapubic cystotomy and drainage are usually indicated. If the posterior urethra is torn, perineal exploration and drainage best meet the local requirements. If the urinary infiltration has gone on to extensive infection, widespread drainage is indicated.

Injury of the Small Intestines. It is practically impossible to know, before operation, whether small intestine is injured or not. Any penetrating wound below the level of the transverse colon is apt to penetrate some portion of it. When the abdomen is opened the small intestines should be inspected in an orderly manner, beginning either at the ileocecal junction or at the first portion of the jejunum. The whole circumference should be inspected and especial care given to the mesenteric border because a perforation here may be easily overlooked. The intestine should be delivered in short sections, examined and returned, then another short section inspected, until the entire small intestine has been investigated. It is shocking to eviscerate the patient and unwise to pull over the coils of intestine in a disorderly manner.

If a hole is found, cover that loop with moist warm gauze, hold the gauze in place with an intestinal clamp, and continue examining the intestine until all the openings are located. Sometimes there are multiple openings, close together, with mesenteric damage. Resection may be the safest method of handling such a section of damaged gut. If one sutures the openings as they are exposed, much valuable time may be lost in suturing openings in intestine that must be resected. Unless a painstaking and orderly examination is made, perforations may be overlooked.

Any widespread injury to mesentery will so seriously interfere with the blood supply as to make resection imperative and multiple perforations placed close together are best treated by removing that section of gut. About one-fourth of all penetrating wounds of the abdomen will injure the small intestine.

Large Intestines. Multiple openings are not very common and the mesentery is not so often seriously damaged. Injuries of the transverse colon, because of its position and the arrangement of its mesentery, more nearly approach in type injuries of the small intestine than do those of either the ascending or descending colon. Colon injuries are very serious, however, for several reasons; the gut is heavily infected and perforation in the post-peritoneal portion of the gut is apt to be overlooked. When found it is difficult of successful closure, because there is no peritoneal coat to seal the closure. It is nearly always necessary to drain and drainage material in close proximity to suture lines is apt to promote non-union, unless the closure can be well covered with peritoneum or tags of fat or omentum. The large intestine is injured nearly as often as the small, especially in stab wounds along the lateral areas of the abdomen.

Duodenum. Wounds of the duodenum are not common. In examining the upper abdomen it is a simple matter to explore the first portion of the duodenum, but a considerable part of the duodenum is behind the peritoneum and perforation or laceration may be overlooked. Should it be found, it requires careful suturing, because of its lack of peritoneum and because postoperative infection of the tissues behind the peritoneum creates a serious hazard.

Stomach. Injuries of the stomach comprise about 10 per cent of abdominal wounds. Operative treatment may be simple or difficult depending chiefly upon the location of the injury. Perforations or lacerations along either curvature may be difficult to close and injury high up near the esophagus is not easily exposed. The most serious problem in stomach injury, however, is to be sure that there is no injury of the posterior wall of the stomach or of the pancreas or the duodenum.

Injuries of the stomach promptly and properly closed give a good prognosis, because the stomach is large and thick-walled and ample closure can usually be gotten without risk of leakage or narrowing of its lumen. While the contents of the stomach may soil the peritoneal cavity, infection is not usually fatal until after considerable time has been lost between injury and operation. A great deal depends upon whether the stomach is full or empty at the time of mishap.

Liver. Injuries to the liver are often serious. They comprise about one-fifth of abdominal wounds. Because of the great size and extent of this organ it should be constantly kept in mind in all injuries of the upper abdomen or lower chest. When it is lacerated or incised two entirely different groups of symptoms may develop; those due to hemorrhage and, later on, those caused by infection. If the wound is a small one, no very serious damage may be done, but in most instances it is impossible to know with any exactness just what is happening until the abdomen is open.

Penetrating or perforating wounds in the right upper abdomen in the immediate vicinity of the liver may be waited on, if there are no symptoms of hemorrhage. This is especially true if several hours have elapsed since the injury. A proper roentgenographic examination is often very helpful. Penetrating wounds of the chest, low down on the right side often injure the liver. If the missile is above the level of the intestines and well to the right side and there is little evidence of abdominal bleeding or infection, many surgeons treat these patients expectantly. If the wound is a stabbing one, bleeding is apt to be more profuse. Because the liver is an excreting gland any considerable amount of bile that escapes into the peritoneal cavity mixed with blood is a menace, creating a disturbing risk. Unless the indications for waiting are clear-cut, operation is the safer procedure.

There is another type of injury that may be overlooked with very great risk to the patient. This is subcutaneous laceration of the liver with active bleeding

into the peritoneal cavity. Here, operation should be done as soon as there is any grave suspicion of such an injury, depending upon the kind of trauma suffered by the patient and symptoms of advancing hemorrhage and shock.

Most of these lacerations are found on the convex surface of the liver just under the upper right abdominal wall. Tenderness over this region with a history of a fall, blow or compression, together with symptoms of bleeding and shock should call for operation as soon as possible. To wait is to court disaster. Most of the blood in the liver is under a relatively low pressure and the bleeding is not arterial, except to the extent that branches of the hepatic artery are opened.

If the laceration is easily reached—that is, if it is in the left lobe or in the under surface of the right lobe or along the edge of the liver just under the costal margin, bleeding may be controlled by suturing the liver with long, round, curved needles passing them through the liver eye first. The sutures must be carefully tied, as tight sutures may cut through the liver substance.

If, however, the laceration is high up on the convex surface under the lower ribs, a great deal of handling may be required to suture such an injury. It is often better surgery to thrust the fingers of one hand between liver and abdominal wall, locate the diaphragm and make an intercostal incision as nearly as possible over the laceration, taking care not to open the costophrenic angle of the pleura. Through this incision one may pass a long, gauze pack, using the fingers of the hand above the liver to tampon firmly such a pack into the laceration and between the wall of the abdomen and upper surface of the liver. I have used this method many times with success. It can be quickly done. The gauze is well away from the intestines. It is anchored in place by the opening between the ribs and later can be gradually loosened and finally removed. A single long pack should be used and the outer end sutured securely to the skin edges. Whether or not a dependent drain should be placed in the abdomen depends upon how dry the peritoneum can be gotten, how large the liver laceration is and the time factor. Such drainage may be necessary, but it always creates the possibility of obstruction.

Spleen. Wounds of the spleen are not so frequent as are those of the liver. They are always serious, however, because the blood supply is abundant and the pressure in the arteries is high. Loss of blood is the outstanding risk and causes most of the symptoms. Laceration of the spleen from concussion or compression is not uncommon. Tenderness may be present, but the spleen is well tucked away below the left diaphragm and concealed hemorrhage may go on without suspecting that the spleen is lacerated. There is no way to make a definite diagnosis before operation and when laceration of the spleen is suspected operation is urgently indicated.

At operation, some clotting may be found in the neighborhood of the spleen. One hand should be thrust between spleen and diaphragm and the spleen rapidly and gently pulled forward and to the right. If bleeding is active it can often be controlled temporarily by grasping the hilum. The space between the spleen and diaphragm should be firmly packed with gauze and with the fingers of one hand controlling the hilum, the spleen may be examined to determine the amount of damage. If there is found a clear-cut laceration, not very extensive, and the spleen is not soft and pulpy, bleeding may be controlled by sutures placed with a long, curved, round needle and tied gently and slowly, but securely. If this can be done it will save time and lessen shock. Most lacerated spleens, however, are best removed, because the lacerations are apt to be multiple and irregular, the spleen pulpy in consistency and the risk of recurrent hemorrhage is considerable.

Pancreas. Sometimes an opening in the posterior wall of the stomach is overlooked

or not securely sutured, complicated by injury to the pancreas. The spilling of pancreatic juice is very damaging. There are two chief approaches to this region—cephalad to the stomach through the hepatogastric ligament (gastrohepatic omentum) or caudal to the stomach through the gastrocolic omentum. The lower approach gives the better exposure; the upper approach is, perhaps, safer because drainage above the stomach is less apt to allow leakage into the general peritoneal cavity and, therefore, localizes infection. The farther drainage can be placed from the region occupied by the small intestines, the less the likelihood of postoperative paralytic ileus and mechanical obstruction.

The pancreas is being brought more and more within the field of abdominal surgery and in an early case of pancreatic injury it may be wise to expose the pancreas, control the bleeding from it and repair the damage by suturing, remembering always the relationship of the head of the pancreas to the duodenum and common bile duct and the body and tail of the pancreas to the splenic vessels. All other branches of the celiac axis are to be avoided, especially the middle colic artery. If operation follows promptly after injury and the damage to pancreas can be securely repaired, a small drain is all that is required. If, however, a number of hours have passed, there is apt to be widespread bleeding and escape of pancreatic secretion. Drainage here should be ample.

Wounds of the Kidney. About one-half of the injuries to the kidney are uncomplicated, but because of the nearness of the right kidney to the liver and of the left kidney to the spleen, many wounds of the kidney are associated with injuries to other structures. If the renal artery or vein is opened, bleeding is apt to be fatal. If the kidney itself is lacerated or penetrated the bleeding may be confined to the perinephritic space, it may force its way behind the peritoneum or if there is a communication between kidney region and peritoneum, there may be free blood in the peritoneal cavity. In many instances there will be blood in the urine and sometimes, though not often, hemorrhage into the bladder may be alarming. If the pelvis of the kidney or ureter is opened at the same time, urinary infiltration and serious infection will occur.

Most uncomplicated injuries of the kidney are not as severe as this. There may be concussion or compression and sometimes the kidney is damaged by a penetrating wound. There may be a moderate amount of bleeding and these less serious injuries should be treated expectantly. Later on, drainage of a perinephritic infection may be necessary or the bladder may require irrigation to clear it of clots.

If the kidney is injured as a complication of some intraperitoneal trauma requiring laparotomy, it is usually not necessary to expose it. If it is found exposed during the intra-abdominal operation because of a communicating wound, it cannot be ignored. It must be dealt with as conditions indicate. The least that should be done is to drain the kidney region through the back and close the opening in the peritoneum as well as conditions and time will permit.

If the kidney is lacerated it may be removed through the abdominal incision. This is, perhaps, the better procedure if the posterior peritoneum is torn. In approaching an injured kidney through the back a good exposure should be made in order to deal promptly with hemorrhage from the kidney that may be encountered as soon as the pressure around the kidney is removed.

*Wounds of the Chest and Abdomen.** "Operation is usually indicated in these injuries: the expectant method can seldom be followed. If a bullet enters the abdomen high up on the right side and passes upward through the liver and diaphragm

* The section which follows is quoted from Shipley, Arthur M. Injuries of the Chest. In Lewis' Practice of Surgery, Vol. IV, Chapter X. W. F. Prior Co., Hagerstown, Md.

into the lung, with no evidence of bleeding into the peritoneal cavity, the patient may be treated without operation and recovery is the rule. One must be quite sure that the entire course of the bullet in the abdomen is through the liver and that there is no large abdominal hemorrhage.

"If a bullet penetrates the right thorax low down and passes through the right dome of the diaphragm, injuring the liver, it is fairly safe to wait, provided there is no large hemorrhage into the peritoneal cavity and provided the course of the bullet is within the limits of the liver. With these two exceptions, immediate operation is indicated when a knife blade or missile penetrates both chest and abdomen. If concussion or compression shows symptoms of abdominal injury laparotomy should be done. When there is evidence of rupture of the left half of the diaphragm, operation is indicated.

"Three different approaches are available and the kind of injury governs the choice of procedure. Thoracotomy alone, laparotomy alone, or both thoracotomy and laparotomy may be done. If both chest and abdomen are to be explored one incision may be made running parallel with the ribs and exposing the lower thorax and upper abdomen. Sometimes it is wiser to open the chest and abdomen through separate incisions. This is true when a missile penetrates into the lower abdomen after perforating the chest. In sucking wounds of the chest associated with penetration of the abdomen, operation on both chest and abdomen is imperative; the chest should be operated on first.

"*Combined Wound of Abdomen and Chest on the Right Side.* An incision across the diaphragm parallel with the ribs gives a good exposure of the chest, diaphragm and right lobe of the liver. Repair of the lung may be done, the opening in the diaphragm closed, and after drying the pleura the chest be closed tightly. Bleeding from the liver should be controlled and the abdomen closed. If bleeding from the liver cannot be controlled by suture, the wound should be tamponed with gauze, the ends of which should be brought out through the abdominal wall below the diaphragm. Suturing the pleura to the diaphragm may help to close the pleura.

"If Roentgen ray examination or wound of exit shows that the missile penetrated the abdomen below the liver, laparotomy should be done first and it may seem wiser not to open the thorax. If the thorax requires exploration, thoracotomy should be done through a separate incision.

"*Combined Wound of Abdomen and Chest on the Left Side.* If the combined wound is on the left side there is much greater likelihood of serious abdominal injury. The stomach, colon and spleen are close to the diaphragm and these structures are apt to be injured.

"If the injury is confined to the chest and upper left abdomen thoracotomy may be done, the wound in the diaphragm enlarged, injury to the stomach, spleen or colon repaired and the opening in the diaphragm closed. After repairing any injury to the intrathoracic structures, the pleura should be emptied of blood and the wound in the chest wall should be tightly closed.

"A better procedure is to open both the lower chest and upper abdomen by a long incision parallel to the ribs extending from the anterior axillary line across the diaphragm and through the upper abdominal wall. This gives a better exposure of the contents of the upper abdomen, makes repair easier and allows drainage of the abdomen if this is necessary. Whether the abdomen is drained or not, the chest should be closed without drainage.

"If the lower abdomen is penetrated, laparotomy should be done first and any abdominal damage repaired. Palpation of the diaphragm from below will indicate whether there is any large opening in it or any hernia through it; in the absence of both, it might be considered wise not to open the chest. On the other hand, if there is a large rent in the diaphragm, with or without protrusion through it, thoracot-

omy should be done through a separate incision, as it is much easier to repair the diaphragm from above and at the same time repair any damage within the chest, as well as empty the pleura of blood."

Statistical Summary. Over a fifteen year period ending September 1, 1938, there were admitted to the University Hospital, Baltimore, 205 patients with some type of abdominal mishap due to trauma. Of these, sixty-nine died, giving a mortality of 33.65 per cent.

One hundred seventy-two were operated on immediately after admission or as soon thereafter as operation could be performed. There are a number of self evident factors that may delay operation for a short time. Of the thirty-three patients not operated on, ten were moribund on admission and died before any operative procedure could be undertaken. Seven were diagnosed as injury to one kidney and were treated successfully without operation. The remaining sixteen patients had exploration of the wound done in the accident room where the injury was found to be confined to the abdominal wall. They were admitted to the hospital for observation or because of some other injury.

Of the 172 patients operated on, sixty-six had gunshot wounds and fifty had wounds caused by stabbing. The remaining fifty-six cases which included both subcutaneous and penetrating wounds were due to a variety of causes, such as crushing injuries or concussion due to falls, blows, automobile accidents or industrial mishaps. Several patients were injured by goring; trauma due to instrumentation was the cause of injury in a small group. In accordance with the classification given earlier in this paper these injuries were divided as follows:

1. Open wounds that do not penetrate the peritoneum	43
A. Wounds of the abdominal wall	18
B. Retroperitoneal injuries	18
C. Wounds of extraperitoneal pelvic structures	7
2. Penetrating or perforating wounds opening the peritoneum	87
3. Injury of any abdominal viscus without penetration or perforation of the skin	42

It is pertinent to call attention to the relative frequency of this last group, a type of injury easily overlooked.

There were twenty-three patients in whom two or more viscera were injured. Consideration of this group is pertinent also, as many surgeons can remember being so occupied with one injury as to fail to locate a second or third one. This group showed a higher mortality than the whole number, as twelve of the twenty-three died. There were thirteen patients who had a combined thoracic and abdominal injury. Most of these were gunshot wounds and the opening in the diaphragm was small. Eight of these thirteen died.

In thirty-five instances the liver was injured. It is interesting to note that in two cases the gall-bladder was torn away from the liver, necessitating cholecystectomy. Both of these patients recovered. Shock of varying degree was present in 33 per cent of thirty-five patients. In eighteen of the cases the operator was able to suture the liver with six deaths. In the remaining fifteen cases gauze packing was used, with eleven deaths. These figures are very expressive, but it should be remembered that in most instances the patient was so shocked that packing was thought to offer the only chance. At operation all patients with liver injury were drained except three. These three recovered, but they were patients without severe laceration. It is the belief in this clinic that injuries of the liver call for drainage.

There were thirty-one patients in whom the jejunum or ileum was opened and in twenty-three of these the openings were multiple. In many of these patients, seen early, there was little indication that the intestine was injured, beyond the evidence of penetration of the peritoneal cavity.

Shock is a misleading symptom in penetrating wounds of the intestine. If seen within a short time following injury, shock may be quite evident and is often psychic. The patient will often recover from this and seem much improved for a

longer or shorter period until hemorrhage or infection develops. Pain is an inconstant symptom and not reliable. Muscle rigidity and tenderness were often absent in the early cases.

In twenty-one of the thirty-one cases the openings were closed without resection, with eight deaths. Resection was done in ten cases with six deaths. In twenty-seven of these patients the peritoneal cavity was drained, with thirteen deaths. Four patients were not drained, and one death occurred in this group.

Injury to the spleen was encountered eighteen times. Shock was the predominant symptom and was present in thirteen cases. In one instance of subcutaneous laceration of the spleen, shock was not present until twenty-four hours after injury. Of these eighteen patients, the spleen was sutured in four, with one death; splenectomy was done thirteen times, with four deaths; and in one moribund case packing was attempted with a fatal outcome. Five of these patients were drained, with four deaths. Only two deaths occurred in the cases that were not drained. Auto transfusion was performed in two of these cases with recovery in both instances.

The stomach was penetrated fourteen times, with five deaths. In two of these the pancreas was injured also; both patients died. The third injury of the pancreas in this group of cases was a subcutaneous one. This patient was admitted three days after injury with a localized infection surrounding the pancreas, which was drained with recovery.

There were twelve intraperitoneal penetrating wounds of the large intestine; ten were sutured, with five deaths. Two were resected and both died. Ten of these cases were drained. There were two that were not drained, with recovery.

There were twelve patients with intraperitoneal hemorrhage, without injury of any viscus. The bleeding came, in each instance, chiefly from a severed artery. The mortality depended upon the vessel injured and the amount of blood lost. In one patient the middle colic artery was severed and in another the superior mesenteric artery was divided. Laceration of the mesentery of the small intestine was a very serious injury. In five such cases there were three deaths.

The urinary bladder was penetrated fourteen times. These were, in most instances, accompanied by injury to the bony pelvis. The diagnosis was made in each case by the history of injury in the bladder region; the presence of surgical shock; inability of the patient to void; and the obtaining of blood on catheterization. In eight cases the opening was an intraperitoneal one. These were treated by repair of the bladder and drainage. Seven of these eight patients died. In six of these cases the injury was extraperitoneal. They were treated by suprapubic drainage, with no mortality. There was one case of extraperitoneal penetration of the rectum. This patient recovered.

Of the retroperitoneal injuries, those of the kidney were the most frequent. There were seventeen such cases. In seven instances mentioned above the injury was not believed to be a severe one and they were treated expectantly. In ten patients there was evidence of extensive laceration. In five of these nephrectomy was done, with no mortality. An attempt was made to control kidney bleeding by packing in three cases, but all three died. In two cases the kidney was sutured, with one recovery and one death.

Postperitoneal penetration of the colon was found in two patients. Both were sutured and drained and both recovered. There were two patients with postperitoneal hemorrhage in whom no attempt was made to locate the bleeding vessel. Both recovered. There was one postperitoneal perforation of the duodenum. This patient suffered from multiple injuries and died.

TRAUMATIC APPENDICITIS

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THE relationship between injury and disease has assumed increasing importance in the administration of workmen's compensation laws. The original legislation was concerned with the more obvious effects of accidental injuries such as fracture, amputation, ankylosis and paralysis. These laws, however, have been liberally construed to include disease produced or aggravated by injury as a proper basis for benefits. The pathologic fracture occurring in a sarcoma of the femur from ordinary muscular effort is considered in the same category with the compound fracture sustained by a fall from a height. The occurrence of hemoptysis in a tuberculous individual after mild effort is considered on a par with pulmonary hemorrhage following fracture of the ribs from a compressive injury of the chest. The fit that an epileptic may take on the floor is given equal legal weight to a seizure resulting in a fall into a tank with subsequent drowning or death from poisonous fumes.

These few examples would indicate that there is a trend in compensation practice to extend its benefits beyond its original purpose. Although designed as an actuarial scheme to provide benefits for reduced working capacity resulting from industrial accidents, compensation legislation has been modified in practice into a sickness insurance scheme with far reaching implications.

This perversion has occurred without the deliberate intent of any group. It has resulted in large measure from the medical opinion that forms the basis of legal decisions.

This medical opinion is not at all consistent. There is, for example, a small group the members of which believe that trauma can actually produce disease and frequently produce a characteristic form of the disease. A second group admits the possibility of trauma aggravating preëxisting disease but denies its influence in initiating disease. A third group denies any relationship between injury and disease. This threefold opinion may be seen in the prevailing attitude concerning the relationship between trauma and appendicitis.

The first group maintains that there is a definite causal connection between trauma and the onset of appendicitis. Liniger[1] believes that a direct and severe contusion of the abdomen may injure a healthy appendix and initiate appendicitis. He does not believe that a physical exertion such as lifting could be a cause.

Wilhelm[2] feels that it is more difficulty to deny the relationship than to affirm it and cites two cases to support his opinion. The first case was that of a chauffeur, age 25, who was injured in an auto accident. Six months later he developed an attack of acute appendicitis. At operation, a hematoma about the size of a lemon was found and within it a gangrenous appendix. Wilhelm concludes that this condition was related to the injury six months previously. The second case was that of a boy of 15 who was struck in the abdomen. Five days later at operation, he showed a definite hemorrhagic appendix.

Sabawala[3] reports the perforation of an otherwise healthy appendix immediately following a horse kick.

While the second group is reluctant to admit that trauma can cause an appendicitis in a healthy appendix, they are firmly convinced of such an influence in the presence of preëxisting pathology. This position is based on a mechanical concept of pathogenesis emphasized by Luding-

ton.[4] Heretofore traumatic aggravation of a preëxisting chronic appendicitis has been explained by direct traumatization of the appendix or its mesentery. The newer theory holds that the mechanism is that of overdistention of the lumen of the appendix by cecal contents as the result of increased intracecal or intraabdominal pressure. While the healthy appendix with a normal lumen might avoid injury by a free return flow of cecal contents, the presence of previous pathologic conditions, such as adhesions, fibrosis or fecaliths might predispose to obstruction and necrosis. Physical changes in the appendix are quite frequent and give rise to recurrent attacks of pain in the right lower quadrant sometimes associated with nausea and vomiting. While chronic appendicitis as a clinical entity is disputed, the symptoms related to these physical changes are relieved by appendectomy and must be, therefore, considered as preëxisting causes favoring the onset of acute appendicitis.[5]

Bissel[6] believes that acute appendicitis can be caused by forcing a fecalith to impinge tightly in the lumen of the appendix with resulting infarction and necrosis. In fifty cases of so-called traumatic appendicitis Kelly[7] found a history of antedating appendiceal disease. Goldbeck[8] as far back as 1830 reported a perforation of a gangrenous appendix coming on eight days after a fall from a wagon.

Burgess[9] reports the case of a tunnel worker who, for a period of two weeks prior to the onset of symptoms, had been using a pneumatic drill, the butt of which rested in his right iliac fossa. While drilling he suddenly developed a pain in his right lower quadrant which doubled him up. At operation the appendix was found ruptured and the tip amputated.

The relationship between trauma and appendicitis is rejected by the third group largely on statistical grounds. They hold that the occurrence of appendicitis following injury is purely coincidental. Fowler[10] compiled the findings of nineteen investigators. Of 13,496 cases of appendicitis reported, there were only 48 or 0.3 per cent in which a history of injury was noted. Levai[11] of the Hungarian State Insurance office investigated the histories of 1,054 cases of appendicitis. In this series he found only nine patients who gave a history of injury. Of this number, one claimed to have been injured by a shove with a foot; three were struck in the abdomen with a work tool; two fell from a height and three claimed injury from exertion or lifting. None showed any definite abdominal symptoms immediately following injury.

One thousand cases of appendicitis personally studied failed to disclose a single history of injury.[12] There were only three cases in which symptoms occurred just before or during the course of work, yet in none was there any definite history of trauma. The histories follow:

W. S., age 28, optometrist. "At 9 A.M. on date of admission, while at work in an optical office, patient suddenly experienced a sharp pain in the right lower quadrant."

N. G., age 26, meteorologist. "This morning patient had abdominal cramps and felt nauseous. He went to work but felt too ill to continue and visited a doctor who advised an operation."

H. A , age 40, steel worker. "While at work this afternoon had severe pain in abdomen with nausea and constant vomiting."

It is the experience of every general surgeon that workmen are inclined to ascribe their symptoms to their work, which is not to be wondered at since in daily occupations numerous small traumata occur. For this reason the histories in cases of alleged traumatic appendicitis must be analyzed very critically.

In the three cases cited the onset of symptoms was associated chronologically with work. It would have been a simple matter for an injury to have occurred during the course of these symptoms and thus, in the mind of the patient be an adequate explanation or cause.

On the basis of statistical evidence, therefore, the causal relationship between

trauma and appendicitis would seem to be remote.

Legal decisions follow this threefold medical attitude so that we find all three positions taken by the courts. Awards have been made on the theory that the appendicitis was directly caused by a blow or strain. An example of this is the case of Clark vs. Department of Labor and Industries, Washington Supreme Court (11/14/24, 131 Wash. 256, 230 Pac. 133). The claimant was struck in the abdomen by a flying block of wood. Pain and nausea followed almost immediately. She was operated on for appendicitis and the appendix was removed. The Department of Labor and Industries denied compensation. The trial court, however, reversed on appeal, which decision was upheld by the Supreme Court. The court said that although the medical evidence was in conflict, the trial court was justified in reversing the prior decision because the weight of evidence showed that the appendicitis was caused by the blow. The decision was said to be fortified by an article in the Journal of the American Medical Association of May 19, 1923 by Dr. Ludington, which stated that appendicitis may and often is caused by a blow on the abdomen or severe muscular strain.

In Thomseth vs. Shapiro Brothers Launderers and Dry Cleaners (Minn. Supreme Court, 4/24/31, 236 N. W. 311), the deceased was struck in the abdomen by a crank handle, apparently after the truck he was cranking backfired. In a short opinion the court said that the evidence was sufficient to show that deceased was struck as alleged and that the medical evidence was sufficient to support plaintiff's theory that the blow caused acute appendicitis to follow.

In the case of Beach Construction Co. vs. Sullivan (N.J. Supreme Court 7/22/35, 179 A. 847), the claimant was lifting, unassisted a radiator weighing 300 pounds when he experienced a slight blow and felt a sharp stinging pain in the region of the appendix. He fell to the floor. He was operated on the next morning for appendicitis. The Workmen's Compensation Bureau denied compensation for lack of causal relation. The Court of Common Pleas reversed. On appeal the judgment of the Court of Common Pleas was affirmed. Although proof on both sides was submitted the weight of evidence including expert medical testimony supports the findings of the Court of Common Pleas, the Court said.

In the case of McDonough vs. Scott Co. (5/27/16, 3 Cal. I.A.C. Decision 225), a steamfitter's helper, while helping to lift a 350 pound hot water generator, sustained a muscular strain in the abdomen. Although the claimant suffered pain at the time he continued to work that day and for part of the following day. The pain was diagnosed as appendicitis; an operation was performed. It was held that the medical testimony showed that appendicitis could be caused by strain in lifting and that under the circumstances of the present case the appendicitis suffered by applicant could reasonably be traumatic appendicitis caused by strain.

In the case of Giese vs. Thomas J. Downing Sons, Inc. (A.D. Third Dept., 9/29/37, 252 A.D. 712, 298 N.Y. Sup. 858), the deceased, a truck driver, had delivered forty-six cases of empty bottles. On return to the employer's place of business he reported sick and went home. Two days later an operation was performed and a gangrenous appendix removed. He died a week later. The Appellate Division unanimously affirmed the award of the Industrial Board. On what theory such award was made, however, does not clearly appear. The employer's first report of the injury stated the deceased was injured in his regular occupation of loading cases of bottles on trucks, each case averaging about 120 pounds, and that the nature of the injury was a hernia developed from lifting.

In other cases compensation has been denied where the medical testimony in favor of the claimant merely showed that

appendicitis could have been caused by the injury but did not show that it was in fact so caused in the particular case. A case of this kind is Consendal vs. Piggott (Mich. Supreme Court 7/16/25, 231 Mich. 544, 204 N.W. 722). Here the deceased helped to move a stove and while so doing was seized with severe pain in the abdomen. He died after an operation for appendicitis. The medical testimony indicated that appendicitis could be caused by strain, but did not state that in this case it was probably so caused. The Supreme Court said that it was a question of fact which had been resolved against the claimant by the Industrial Board, and that the finding of the Board was binding on the court.

In Tomovich vs. Department of Labor and Industries (Washington Supreme Court 9/5/23, 218 Pac. 197), the claimant fell and struck his abdomen on a small log. He was sent to a hospital suffering from appendicitis. Other complications developed. Compensation was denied. The Court said that the only testimony in the claimant's favor was that appendicitis could have developed from the injury or that the injury could have contributed to his condition. This mere possibility, it was found, was not sufficient in the face of positive and contrary opinions of medical witnesses.

In the case of Tucker vs. Wilson and Co. (Oklahoma, Supreme Court, 8/2/27, 258 Pac. 905), the claimant slipped and fell, allegedly "tearing his side." He was found to have appendicitis. Medical testimony for the claimant indicated his trouble could have been caused by the injury, but was more likely to have been caused by some other source. The Industrial Board denied compensation. On appeal, the decision held that the evidence sustained the Board's findings.

An example of cases involving mere aggravation of preëxisting conditions is that of Fritz vs. Rudy Furnace Co. (Mich. Supreme Court, 6/5/22, 188 N.W. 528). In this case a foundry worker, assisting in pouring molten iron, suffered a wrench when the ladle slipped or caught, at which time he felt a pain in the region of the appendix. At first he was treated as though overcome by heat. Later his condition was diagnosed as appendicitis. During the operation adhesions were found and other evidence of a chronic condition. Compensation was awarded.

In Watkins vs. Brunswick Restaurant (Nebraska, Supreme Court 5/6/32, 242 N.W. 439), a waitress fell, striking her right side, when she stood on a table to reach some overhead shelves. The appendix was removed four days later. Medical testimony was to the effect that she might have had a diseased appendix, and if she did, it might have "lit it up." The evidence was found to sustain the finding that the fall caused the appendicitis. In the course of its opinion the Court said: "And it also appears that while there is a lack of entire harmony by medical authorities in respect thereof, a trauma or blow may cause appendicitis."

In Lindquest vs. Holler (A.D. 3rd Dept., 5/2/17, 164 N.Y. Sup. 906), the deceased, a superintendent of construction of part of a barge canal, fell while climbing out of a prism of the canal, striking his abdomen and causing severe pain. He died of acute peritonitis. No autopsy was performed. It was held that the evidence was sufficient to sustain finding that death resulted from acute peritonitis caused by rupture of the appendix which was diseased at the time of the accident.

In Shadbolt vs. Department of Labor and Industries (Washington Supreme Court, 10/6/22, 209 Pac. 683), compensation was awarded for disability from a diseased appendix which was ruptured by the pressure of blocks of wood which claimant was transferring from a platform.

Lack of causal relation between the appendicitis and the injury was found in the following cases:

Weaver vs. Industrial Commission of Colorado (Supreme Court, 7/3/22, rehearing denied 10/2/22, 209 Pac. 642). In

this case the deceased died from acute appendicitis on March 23, 1917. Some witnesses testified as to a causal connection between the death and burns suffered by the deceased on November 24, 1915. The Commission found against the claimant, and the court held that the findings of the Commission were upheld by the evidence.

In Dube vs. Clayton Brothers, Inc. (10/21/15, 1 Conn. Comp. Commissioners' Decisions, 441), the claimant alleged that he had fallen against the corner of a tank, thereby sustaining a rupture. It developed that he had an attack of appendicitis and the appendix was removed. He had previously had trouble with his appendix. Compensation was denied on the ground that (1) no claim was made within the required period, and (2) that even if he had received a fall as he claimed, the medical evidence was to the effect that it would be highly improbable that such a fall would cause a ruptured appendix or be in any way connected with the appendicitis which the claimant undoubtedly had.

In Chambers vs. Universal Advertising Corporation (A.D. 3rd Dept., 1/12/38, 253 A.D. 854, 1 N.Y. Sup. 2nd 497), a painter fell from a scaffold resulting in a fracture of the pelvis and other bones, and many other injuries. After three months in the hospital appendicitis developed. He was operated upon and died a week later. This was held compensable. The Court said that the medical evidence established that the appendicitis was not the result of the accident, that the deceased would have recovered from the accident were it not for the appendicitis, but that the appendicitis would not have been fatal except for conditions resulting from the accident, and that the accident was a contributing and indirect cause of death.

COMMENT

The distention of the lumen of the appendix by trauma with subsequent impairment of the appendiceal circulation and necrosis of the wall of the appendix is a logical explanation for appendicitis, but one whose validity is not substantiated by a sufficient number of cases. One would certainly expect a larger incidence in the general population. One would furthermore anticipate a higher incidence in those occupations where abdominal injuries and strain are frequent. Although there have been no factual studies of the occupational incidence of appendicitis, it has been my impression from the examination of large numbers of injured workers that laborers who exert a great deal of back and abdominal strain, manifest very little, if any, appendicitis. The same holds true of others who are subject to severe external trauma, as prize-fighters.

There has been no proof to indicate that the phenomena of exaggerated distensibility of the appendix cannot occur by natural means. Ray[13] describes two features of the anatomy of the appendix that predispose to inflammation: it is a narrow blind sac exposed to a stream of fecal material with a high bacterial content; second, its lymphatic character renders it especially susceptible to inflammation similar to the reaction of other lymphocytic tissues in the body. It is not difficult to understand, therefore, how natural peristaltic movements may produce increased flow of the normal fecal stream to and from the appendix. The obstruction of this material may impair the appendiceal circulation, producing passive congestion of the wall, vascular thrombosis with gangrene and perforation without the intervention of trauma.

SUMMARY

For the purpose of arbitrating claims for traumatic appendicitis, the arbitrator must rely on certain criteria or postulates in order to establish a reasonable relationship between the injury and the disease. The position taken here is that it is highly improbable that trauma can initiate appendicitis. Such a condition should be accepted only when the individual case satisfies the following criteria:

1. No symptoms or signs of appendicitis should have existed before the accident and the individual must have been able to work.

2. Severe injury of the abdomen or overexertion must be proved.

3. There must be immediate evidence of illness which leads to cessation of work at once. An interval of two or three days without any symptom and the full ability to work makes the asserted relationship improbable.

The relationship between trauma and aggravation of appendicitis should be accepted only when it satisfies the following criteria:

1. The injury must have been severe enough to cause at least a temporary cessation from work.

2. The nature and location of the injury must have been such as could affect the appendix.

3. The symptoms of appendicitis must follow the injury directly.

4. In case of death the autopsy should show that the appendix, through its abnormal position and condition, could have been influenced by the injury.

There is no great legal distinction between initiation and aggravation of disease since compensation is paid in either case. The increasing trend toward compensating disease associated with accidental injuries would indicate a need for redefining workmen's compensation procedure to carry out legislative intent. Shall it be insurance against accident, or shall it be sickness insurance?

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EXAMINATION OF THE INJURED BACK

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BECAUSE of limitation of space this article will, of necessity, be confined to examination of what may be termed "minor injuries of the back." This, in general, covers back cases appearing at the examining room of the hospital or the physician's office soon after disability begins following some type of minor accident at work, on the street, or in the home. The patient thereafter develops soreness severe enough to disable him from his regular vocation to some extent. Another group of cases may also be included; these patients have been disabled for some time and because they do not respond after ordinary care, including physiotherapy or immobilization of the back, they are referred for a more exhaustive diagnosis and appropriate treatment.

It will be necessary to omit a discussion of the history of the occurrence of trauma, although this is of course, of great significance. A complete careful description of the type of accident to which the disability is ascribed may be of utmost importance in distinguishing between, for instance, muscle sprains and myositis or arthritis entirely unrelated to accidents. An exhaustive anamnesis such as is taken in the examination of any patient cannot be described here although essential to proper diagnosis.

The examination may be divided for our purposes into a general examination of the patient and a routine examination of the back itself. This latter includes observation of standing and sitting posture and motions; also locomotion, conduct of the patient in undressing and climbing onto the examining table, and the systematic passive performance of motions in the supine, prone, and side-lying positions, including certain special movement tests.

The general examination of the patient, including the detection of various foci of infection which may be capable of initiating or aggravating low back pain, cannot be detailed here.

While I realize the inadequacy of many procedures used in routine examination of the back, a systematic examination, including a roentgenologic study and certain laboratory work, should enable an experienced physician to make a decision:

1. As to whether any actual physical disability exists based on definite objective findings corresponding to the complaint made by the patient.

2. As to the nature of this actual disability, for instance, whether it is due to sacroiliac, lumbosacral or intervertebral pathology, muscle or ligamentous injury, or a myositis or nontraumatic arthritis.

Some tabulated form for recording each objective finding is necessary for comparison on subsequent examination.

Determination of the exact nature of the disability is of prime importance since upon a definite diagnosis depends the type of management which each case deserves. One is likely early in his experience with the study of injured backs, particularly in compensable cases or those in which some settlement is expected as the result of a public accident, to believe that all patients grossly exaggerate their symptoms or that the complaint of soreness in the back is to a large extent on a neurasthenic and not on a physical basis.

An analysis of the findings on routine examination is made tremendously more difficult by the fact that physical signs produced by the actual painful lesion are in many cases overshadowed by signs produced by spasm, contracture and rigid-

ity of the muscles moving painful joints rather than by the joint pathology itself, or by signs produced by radiation of nerve pain to areas at some distance from the seat of the actual pathology.

Since the greatest number of disabilities involve the lower back, consideration will be given in particular to examination of this part.

PHYSICAL EXAMINATION

The patient must be stripped for the examination. It is valuable in watching him while he is disrobing and dressing again to note how far he is able to flex the spine forward and how much lateral motion he can perform during these operations as compared with how much motion he allows while being examined on the table. Observe whether he sits on one buttock more than the other in disrobing and whether he favors his back while climbing upon the table.

After the patient is stripped he should be examined standing equally on the two feet, as viewed from behind, from either side, and from in front. The height and prominence of each gluteal fold is noted. He is asked to flex each knee and hip standing on the other leg (the well-known "Trendelenburg test"), to observe whether, as in the case of a normal hip, when one limb is raised from the floor the buttock on this side rises perceptibly, or whether it sags, which generally indicates hip joint or sacroiliac pathology.

From the side the fascia lata is palpated to see whether it is more tense than normal and whether it stands out more than its fellow of the opposite side. Its relation to the trochanter is also noted. The degree of the lumbar lordosis is studied in relation to the general body type of the patient; i.e., whether it represents the degree of physiologic lordosis one expects in a tall or asthenic type, or the comparatively slight lordosis of the short wide type of back. In excessive lumbar lordosis it is important to note whether this occurs at or just above the lumbosacral junction or whether the greatest curve is in the lumbar segments higher up. Visceroptosis may be a part of the picture in cases of unusual lumbar lordosis, although it is doubtful whether this condition is alone responsible for lumbar pain due to the pull of the displaced abdominal viscera.[1,2,3]

The existence and degree of sciatic scoliosis or deviation to either side in the lumbar region can be seen. Also one may note whether or not it is compensated higher up to bring the shoulders level, as commonly occurs in sciatic scoliosis of lumbosacral origin.

Inclination of the pelvis, that is, the relation of the plane of the sacrum to the horizontal plane is of interest. It is well to test the height of the crests of the ilia, although this is no absolute criterion of the length of the two legs. The legs should be measured from the anterior superior spine to the internal malleoli with the patient standing squarely and the toes pointing forward. If one leg is found to be shorter than the other, this may account for a lateral tipping of the pelvis, causing a compensatory curve in the lumbar spine and not a true sciatic scoliosis. If a patient with a short leg stands with a suitable block under the foot one can observe whether the lumbar lordosis flattens out by this maneuver. If so, it is not spastic but postural.

If the height of the two shoulders is unequal the chances are ten to one that we are dealing with a rotary scoliosis and not a pure sciatic, and therefore perhaps traumatic, scoliosis of the lumbar region. Comparison of the amount of lordosis of the lumbar spine and also, when present, the degree of sciatic scoliosis in the standing as compared with the sitting position is of utmost importance. Little or no difference indicates spasm of the lumbar muscles rather than a developmental defect of the spine, in which one expects no muscle spasm to maintain the standing lordosis or lateral deviation in the sitting position.

If, in the standing position, the sciatic scoliosis patient is unable to extend the hip joint on the concave side completely there is an inference that the hip joint or the sacroiliac joint of this side is affected. If the deviation of the spine is practically unaffected by changing from the standing to the sitting position it is probable that the hip joint is not at fault.

In walking the patient is likely to drag the foot and hold the leg in slight abduction in any case of sciatic scoliosis. In sacroiliac soreness he is prone to walk with the hip and knee slightly flexed, never extending these joints completely. Ordinarily the patient makes more complaint of pain in the lumbosacral or sacroiliac region on walking down than on walking upstairs. When starting to walk the patient with soreness in either of these regions may step off with the leg of the affected side rather than with that of the sound side. When asked to stand on one foot without the aid of the other, the patient with sacroiliac soreness has difficulty in standing on the leg of the affected side. If a unilateral lumbosacral soreness is of any great severity he will also have pain on the affected side while trying to stand on the good leg, a point that a patient who is malingering will never make.

Flexion of the lumbar spine is now studied to see whether the spine deviates to one side in forward flexion, whether there is any lag or limitation of motion at any level of the lumbar region, or whether some level moves only after complete flexion of the other segments has been attained. Forward flexion of the spine is likely to be more limited and painful at an earlier degree of motion in sacroiliac soreness than is such motion in the sitting position. This may be due to the pull of spastic hamstring muscles placing a rotary stress on the sacroiliac joint in the standing position, which is not exerted when the patient is sitting.

The articulations of the lumbar spine are more on the stretch in the sitting than in the standing position, with the exception of the lumbosacral joints, so that one may expect freer forward motion in the standing position with an arthritis of the upper lumbar joints, whereas this may not be true if the lumbosacral joint is affected. If a patient with low back soreness is asked to flex the hip, keeping the knee straight, he will invariably flex the knee when he attempts to perform this motion.

After having the patient flex his spine it is important to note where the motion first takes place as he begins to rise to the standing position again. Soreness at any level is apt to be expressed by a lag of motion or failure of the joints here to move until complete extension has occurred at the other levels.

Rotary motion is less satisfactorily studied in the standing than in the sitting position, because while standing rotation of the pelvis accompanies a turning motion of the spine and spastic thigh muscles may limit the spine motion in this position and obscure the findings.

Examination in the Sitting Position. In this position the normal standing lordosis should be practically flattened out unless it is maintained by lumbar muscle spasm or by mechanical interference due to a change in the spine. "Sitting kyphosis" indicates weakness of lumbar muscle development, an involuntary effort to prevent torsion on a tender sacroiliac joint, or the posterior prolapse of an intervertebral disc which may slide back into place on forward flexion and again herniate on assuming the position of lordosis. I have never seen a sitting kyphosis due to painful impingement of the spinous processes on each other, although this has been described. The type of lordosis produced by spondylolisthesis causes a very evident defect of flattening when the patient sits. In a bilaterally sacralized fifth lumbar vertebra the distance between the lower ribs and the ilia may be less than one would expect for the particular body type of the patient being examined.

The alignment of the spinous processes should be determined by palpation. Palpa-

tion of the lumbar muscles for fibrosis, contracture, or involuntary defense reactions (like Hartman's defense reaction in the abdominal wall in cases of intraperitoneal pathology) can better be accomplished with the patient in the prone position than while sitting. However, it is valuable to compare the amount of change appreciated by the examiner's fingers in standing and in the sitting positions.

While sciatic scoliosis is generally less noticeable in the sitting than the standing position it may be of the same degree in the two positions if a spasm of the erector spinae group and quadratus lumborum is quite marked. To determine whether this deviation is due to spasm on the concave side or muscle relaxation on the opposite side the patient can be lifted by his head, during which maneuver the deviation will disappear if due to a relaxation of the opposite lumbar muscles.

Heavy percussion on the patient's head elicits a complaint of sharp pain in an involved joint of the dorsal or lumbar spine often quite well localized. Conversely, lifting a patient by his head relieves this pain.

Active flexion should affect all parts of the lumbar spine equally so that a true curve is produced, although physiologically there is slightly less motion at the lumbosacral joints than at those above. The amount of deviation to one side or the amount of lag or lack of motion at any level is now studied as compared with findings in the standing position. On forward flexion the patient will stoop toward the side of spastic erector spinae muscles and very commonly away from the side of a painful or compressed fifth lumbar nerve root or posterior disc herniation.

Extension of the lumbar spine is not well accomplished by the patient without the assistance from the examiner in the form of support for the dorsal spine by placing the hands across the patient's back. Attention is directed to the degree of painless extension as compared to flexion. Pain of spondylolisthesis, spondylolysis or ligamentum flavum pathology may be relieved by hyperextension, and pain of lumbar and sacroiliac arthritis and of posterior disc prolapse is much aggravated.

The normal amplitude of rotation in the lumbar spine is very limited at any one joint,[4,5] the apparently great flexibility on examination being due to a combination of flexion in the lumbar region and rotation in the thoracic region. Active rotation is mainly accomplished by contraction of the quadratus lumborum and abdominal muscles on the side opposite the direction toward which the trunk turns on the pelvis.

Voluntary and assisted rotations of the lumbar spine are next produced. The degree of painless rotation is recorded so that this may be compared with the amount of rotation obtained on passive motion while the patient is in the supine or side-lying position. Soreness in the upper lumbar and lower dorsal region is best detected by these rotations in the sitting position, while the findings in regard to lower lumbar pain can only be interpreted after rotations in other positions have been performed. After rotations in the erect sitting position these should be repeated with the patient leaning backward to compare the range of painless motion. This is important in deciding whether an impinging fifth lumbar transverse process or sacralized fifth is painful, also for the study of a suspected posterior disc prolapse.

Lateral motion in the lumbar region is confined to an arc of not over 40 degrees.[1] If a sciatic scoliosis exists and is due to joint or muscle injury, limitation of lateral motion is toward the unaffected side, and on attempting this motion the pelvis tends to rotate toward the unaffected side. Comparison of the arcs of active and passive lateral motion is now recorded.

Examination in the Supine Position. A low examining table, 20 to 24 inches in height, is preferable to the ordinary office type of physician's table, and it should be long enough for the patient to lie at full length. An 8 inch stool is used

by the patient in mounting the examining table. Yergason[6] suggests that when a patient steps up briskly on a table using the unaffected leg his attention is focused on this leg and he does not realize that a considerable shearing force is directed toward the opposite sacroiliac joint, while a patient with marked soreness here cannot step up on the good leg without some complaint of pain.

If the lumbar region is uniformly painful or there is a disabling condition at the lumbosacral junction, the patient will climb upon the couch in a prone position and then roll over onto his back with some difficulty. When lying comfortably on his back the patient of the slender flexible type of body normally presents less lordosis than in the standing position. Painful lumbosacral pathology or spastic lumbar muscles will cause him to maintain an unusual degree of lordosis.[7] A patient with coccygodynia will complain when lying flat on the table. When a patient mounts on the table by first sitting on the edge and then turning himself around, he may support the leg on the side of a unilateral sacroiliac lesion with his hands. If he can manage this leg without difficulty and without the assistance of his hands one does not expect to find the straight leg raising test positive until a considerable degree of flexion has been attained at the hip. A patient with lumbosacral or sacroiliac soreness may refuse to lie flat on the table on his back and can lie more comfortably rotated slightly toward the painless side with the opposite knee and hip somewhat flexed. Insistence on lying with one hip and knee flexed is more suggestive of a sacroiliac than a lumbosacral soreness.

The patient is told to flex the hip and knees. In lumbosacral soreness the persistent lumbar lordosis does not change. In sacroiliac soreness one expects it to be effaced.

In a patient with slight sciatic scoliosis from an irritative lumbosacral lesion this is less painful in the supine position and occasionally after slight manipulation by the examiner the lateral tilt of the pelvis may entirely disappear, indicating the trivial nature of the pathology producing the muscle spasm.

Hip Motion. With the knee flexed to right angles, motions of the hip joint may be studied without the transmission of tension to the sacroiliac or lumbosacral joint and spasm of the hamstrings relaxed. A complaint of painful internal or external rotation of the hip in this position leads to a suspicion of pathology in the hip joint itself.

Lateral and rotary motions of the lumbar spine can easily be performed by the examiner after flexing the hips to right angles with the knees at 60 degrees and passing the right forearm under the patient's knees. One expects passive lateral motion of the lumbar joints to be performed through a larger arc without complaint of pain than in the standing or sitting positions if the pathology is lumbosacral.

The classical test of compression of the two anterior superior spines of the ilia toward each other and separation from each other can be performed in this position. These maneuvers are not of value in the average sacroiliac case, and are painful only if sacroiliac soreness is extreme.

It seems probable to me that all strains of the sacroiliac joints and more particularly of the posterior sacroiliac ligaments, which I believe to be commonly the cause of the so-called sacroiliac soreness after trivial injuries, are rotary in nature. For example, when a patient falls, landing on his feet with the spine erect, a counterclockwise rotary strain as viewed from the right is placed upon the right iliac joint by the right ilium or conversely the upper part of the sacrum rotates downward and forward on the right ilium at the right sacroiliac joint. If a patient sitting in an automobile with the knees, hips and lumbar spine flexed sustains a jarring trauma, the right ilium rotates in a clockwise manner as viewed from the right side at the right sacroiliac joint, or conversely the upper

part of the sacrum rotates backward on the right ilium.

Therefore tests for sacroiliac soreness due to trauma must be directed toward reproducing the type of torsion strain sustained in the injury.

The straight leg-raising sign[8] applied to the right leg reproduces a counterclockwise rotation of the right ilium on the sacroiliac joints as viewed from the right side, or conversely a forward motion of the upper part of the sacrum on the right ilium. This straight leg-raising sign is not the original Lasègue sign but is attributed to him. The original sign was elicited by first flexing the hip and knee and then forcibly extending the knee.

The difficulty in interpreting the findings of the straight leg-raising test depends upon the fact that this is a quantitative test, stress placed upon various structures depending upon the angle of flexion of the hip. Approximately the first 20 degrees of this hip flexion may elicit pathology in the hip joints. As the motion continues and a pulling is exerted on the ischial tuberosity by the hamstring muscles a rotary strain is placed on the sacroiliac joints as above described, and at about 80 or 90 degrees of flexion, torsion begins to be strongly transmitted through the lumbosacral joint of this side. After this point even soreness in the opposite lumbosacral joint will begin to give painful symptoms as the stress is transmitted here. At the very beginning of leg-raising the sciatic nerve trunk is stretched, giving complaints down the sciatic distribution. There is considerable confusion regarding findings in sciatic root pathology. Throckmorton[9] believes this test is valuable in determining sensitiveness of the opposite sciatic nerve, since when the leg is raised to approximately a right angle, the gluteal muscles of the opposite side contract in order to force the heel of the resting leg against the table. If the opposite sciatic nerve or piriformis muscle is sensitive or spastic, gluteal and piriformis contractions may produce sciatic pain here. If the straight leg test is applied until pain begins, a sudden dorsal flexion of the ankle (Braggard's sign) will greatly aggravate sciatic pain but will leave lumbosacral and sacroiliac pain unaffected.

Mennell[10] has contributed an important addition to this test, which I apply routinely. If the straight leg is raised until pain begins and then the ilium of that side is pushed down against the table by the examiner's left hand, relieving the pain, it is obvious that the pain is sacroiliac and not referable to the hip joint.

Laguère's sign, performed by flexion of the hip and abducting and rotating it outward, forcing the femoral head against the anterior part of the capsule, is positive in hip joint conditions but leaves the sacroiliac joint pain unaffected, and very little strain is put by this test on a lumbosacral articulation. It is used to distinguish psoas spasm and hip joint pathology from sacroiliac and lumbosacral pathology. This test puts no strain on the fifth lumbar nerve root or funiculus.

Gaenslen's test[11] applied, for instance, to the right leg, is a means of eliciting soreness in the right sacroiliac joint by a clockwise torsion of this joint. In the supine position the left knee and hip are completely flexed. The patient locks the lumbosacral and left sacroiliac joints by firmly encircling the left knee with his arms. His right hip is then hyperextended over the edge of the table with the knee flexed. The mechanism of rotation of the right sacroiliac joint is easily appreciated. This test can be repeated later in the side-lying position.

The legs are next placed close together with the knees extended and both are raised with one hand of the operator. In this test the lower lumbar spine is moved at a relatively early stage in the maneuver; whereas tension on the sacroiliac joints and the sciatic nerve is at a minimum until the hips are flexed through a considerable range, so that pain may begin in the lumbosacral region as soon as a few degrees of motion are accomplished and through a

smaller range than when either leg is raised separately, and the suspicion of pathology rests here rather than on the sacro-iliacs or the sciatic nerve roots. Hip joint disease, in which the pain is more likely to be referred along the anterior surface of the thigh than elsewhere, can often be distinguished by Patrick's sign, in which the ankle of the affected side is placed on the patella of the opposite outstretched leg and the flexed knee is then pressed downward and outward. In concluding the examination in the supine position deep palpation should be made in each lower quadrant of the abdomen to search for a suspicion of Baer's[12] sacroiliac point of tenderness, described as situated 2 inches from the umbilicus on a line between this and the anterior superior spine.

Examination in the Side-Lying Position. The patient can be examined when lying both on the painful and on the sound side. Presuming that the complaint is directed toward the left side, begin by examining him when he is lying on this side if he will allow this. The hip and knee of this side are completely flexed and the knee grasped by the patient's arms to lock the left hip, sacroiliac and lumbosacral joints, so the manipulations of the sound leg will not be transmitted through to these joints. Flexion of the right straight leg and subsequent extension are compared for range of painless motion with motions of the opposite hip with the patient lying on the right side. After this Ober's test[13] can be performed while the patient is lying on the sound side. The pelvis is held down against the table by the examiner's left hand while his right hand then flexes the knee of the uppermost leg to a right angle and then abducts this thigh through the complete range of motion at the hip and then hyperextends this joint. If a contracture of the fascia latae is present the leg will remain in the abducted position at a degree of abduction proportional to the amount of contraction of this band.

In the side-lying position it is valuable to perform complete passive rotations of the lumbar spine to see how these compare with similar motions in the sitting and supine positions, and lesions referable to any particular part of the thoracic or lumbar spine or the lumbosacral junction can sometimes be definitely localized by the patient's complaints. During these rotations a popping or snapping sound may be produced by stretching periarticular adhesions, and the point of pathology may be determined by the palpating finger.

Examination in the Prone Position. The amount of lateral deviation found is compared with that in the standing and sitting position. Also note the degree to which this distortion can be corrected by pulling down the leg on the convex side. In this position it is possible to produce a clockwise rotary strain on the right sacroiliac joint as viewed from the right and the opposite type of motion and counterclockwise strain on the left sacroiliac joint as viewed from the left without the possibility of this force being transmitted through to the lumbosacral joint. One first makes firm pressure on the lumbosacral region with the left hand anchoring the pelvis to the table while the flexed hip and knee of the side to be studied are forcibly hyperextended.

A hyperextension strain can be placed on the spinal joints by raising the trunk from the couch with the examiner's right arm while counterpressure is made first on the lumbar region with the left hand and then further down on the sacrum as the stretch continues during the elevation of the patient's chest from the table. Observation of the point at which the onset of painful symptoms begins is compared with the position in which extension of the hips produces pain when this motion is continued until the pelvis rises from the table while the chest is held down. The prone position is the best in which to palpate for "rheumatic nodes." Palpating from above downward, one is likely to find them in the regions where muscles are transformed into aponeuroses, as along

the attachments of the erector spinae group to the sacrum or along the upper borders of the ilia. Occasionally they are found about the spines of the scapulae or the outer borders of the erector spinae group. Tenderness along the course of the sciatic nerve may be palpated in this position.

The erector spinae group is one large muscle belly on either side comprised of the spinalis lumborum, the longissimus dorsi and the iliocostalis lumborum, named in order as one travels from the midline and when these are examined manually the three components are rarely distinguished. The short muscles running vertically between the transverse processes and the obliquely situated rotators cannot be identified. A tendency to spasm of these long muscles of one side of the back as compared with the tone of the muscles of the opposite side can best be made out in the prone position. Sometimes a diminution of the actual bulk of the lumbar muscles on one side and a cord-like hardening or fibrosis can be distinguished. I have occasionally been able to palpate the lateral edge of a contracted and spastic quadratus lumborum. The examiner should be able definitely to determine whether the spasm is voluntary or involuntary by repeated examinations. A Hartman's defense reaction can often be detected in spastic and sensitive lumbar muscles. Schade[14] believes that one can estimate the amount of muscle loosening "myogelosen" on the opposite side by palpation, but I have had difficulty in demonstrating this. Repeated examinations of the muscle tone after baking and massage, as pointed out by Lange,[15] may help one to determine whether the change is inflammatory or spastic, since the latter will yield much more readily to physiotherapy than the former. In inflammatory changes it is difficult to distinguish a definitely tender spot in the contracted lumbar muscles, whereas in pure muscle injury with the attendant spasm this can often be done.[16]

In 1928 Yeoman[17] made the first reference to spasm of the piriformis muscle in relation to sciatic pain and coccygodynia. This muscle is relaxed by external, and stretched by internal, rotation of the thigh. In palpating the gluteal muscles for sensitive areas or induration, spasm or contracture, it is difficult to make out tenderness in the piriformis muscle itself. When examined from behind, this muscle is well covered by the glutei. The direction of the muscle belly is approximately from the middle of the sacrum to the upper part of the greater trochanter. Deep palpation should be made along this line. If a rectal examination is performed simultaneously, tenderness in the piriformis may be detected.

Steindler[18] has made an important contribution to the study of low back pain and its relation to musculo-aponeurotic or ligamentous injury by his method of distinguishing pathology here from lesions of the deeper structures. He has selected a group in which he could locate a definite point of tenderness in the sacral triangle near the posterior inferior spine or crest of the ilium, or tissues behind the lumbosacral joints, or in the gluteus maximus and tensor fasciae latae of the greater sciatic notch accompanied by radiation and faulty posture. In these localized pain in identical locations was complained of on leg tests. Following his technique I have injected 5 to 10 cc. of 1 per cent procaine hydrochloride at the point of localized tenderness in an effort to distinguish whether the pathology was local and relieved by such injections. He finds five postulates which must be fulfilled to prove that pain and its radiation are due to pathology in these local structures.

1. Contacts with needle must aggravate the local pain.

2. Contacts with needle must elicit or aggravate the radiation.

3. Procaine infiltration must suppress the local tenderness.

4. Procaine infiltration must suppress radiation.

5. Positive leg tests must disappear.

If superficial tender areas are successfully injected and thereafter the leg signs disappear it is reasonable to assume that in a case of injury the trauma is actually a localized and superficial one.

Examination of the Muscular System. Wasting or fibrillary twitching of muscle groups, especially in the buttocks or legs, should be noted. The circumference of the calves should be measured if there is any diminution in the size of muscle groups here. Mennell[10] makes an important point of testing sensitive muscle areas with faradic current to distinguish between inflammatory and traumatic change, the latter being much less sensitive than the former to faradic stimulation.

The Reflexes. We are interested particularly in the reflexes of the lower extremities. In sciatica the ankle jerk may be diminished or absent. The knee jerk is not affected unless there is a general involvement of the lumbar nerve roots or the lumbar plexus. A positive Babinski, Gordon, Oppenheim, or ankle clonus, indicates a central nervous system origin for any accompanying low back pain.

Rose's[19] gluteal reflex, consisting of an exaggerated fascicular contraction in the gluteus maximus on percussion, if present in sciatica, indicates an involvement of more than the fifth lumbar nerve root.

Laboratory Examination. Routine blood counts and urine examination are part of the study of every back case. In an attempt to distinguish between arthritis and other affections of the spine I have run 200 sedimentation rates. Buckley[20] has found the sedimentation rate markedly increased in infective arthritis. His findings agree approximately with those of Coke[21] and Stangier.[22] I have found no cases with increased sedimentation rate which could not easily be explained on the basis of some other infective process besides back pathology, as for instance, prostatitis or sinus infection or marked pyorrhea. I have not been able to find an increased sedimentation in any case presenting rheumatic or fibrositic nodules in the muscles of the back.

Bittorf[23] has reported eosinophilia in the acute stages of lumbago or myositis in distinction from injuries of the back, but I have not been able to verify these findings by differential counts. General lumbar muscle soreness is sometimes associated with increased uric acid in the blood which is an important consideration in treatment. Apparently however the uric acid may not always be increased, even in very acute cases of gout.[24]

When a blood Wassermann test is positive, antisyphilitic treatment sometimes relieves back soreness and I have been unable to explain this on any basis except that of syphilitic arthritis, although such a diagnosis is debatable.

When a lumbar puncture is performed it is important to try to get below the lumbosacral disc since a posterior herniation here is likely to give symptoms of a partial or complete subarachnoid block by the Queckenstedt test. Xanthochromia may be present. Mixter and Ayer[25] found an elevation of the spinal fluid protein in twenty-two cases where herniation was verified by operation; there was usually less than 200 mg. of protein per 100 c.c. of fluid. Twenty mg. per 100 c.c. of fluid is considered a high normal level by the technique used.

Love and Camp[26] thought that an elevated protein was essential to a diagnosis of a posterior herniation of a disc, but later in eleven cases out of forty-one where the pathology was verified there was a normal protein content.

Love[27] reports a reverse Queckenstedt test, performed by introducing a spinal puncture needle below the lesion and thereafter producing pressure on the terminal dural sac by injecting 1 per cent procaine hydrochloride into the caudal epidural space.

Interpretation of the Findings of the Examination. There may be a triple group of painful symptoms in each individual case. The first is a primary lesion

in joints or periarticular structures, ligaments or muscles. The second is a painful spasm or contracture in muscles to immobilize the original lesion. The third is referred nerve pain, which may be a purely reflex phenomenon or pain localized in the dermatomes supplied by the posterior divisions of the lumbosacral nerves.

Steindler has called attention to the reference of deep pain in a nerve root injury or compression through the "nervus-sinuvertebralis" described by Luschka[28] which is distributed around the intervertebral veins and the basivertebral veins, the pedicles and the interiors of the vertebral bodies.

PHYSICAL FINDINGS OF CERTAIN LOW BACK SYNDROMES

Sciatic Scoliosis. The earlier writers, Charcot[29] and others, describe this clinical syndrome, noting its occurrence in relation to sciatica, hence the term "sciatic scoliosis." A characteristic lateral tilt in the lumbar region without a prominent element of rotation presents a posture which will relieve or mitigate pain-producing lesions. Evidently such a scoliosis is not necessarily traumatic, and four types of pathogenesis have been suggested:

1. A reflex muscular contracture.[30,31]

2. A shifting of the body weight to prevent pressure on painful lesions resulting in a deviation laterally to the sound side.[32,33,34]

3. A spasm of injured muscles on the same side as the muscle or ligamentous strain.

4. An asymmetrical deformity of the lumbar spine, e.g., unilateral sacralization.

The general impression in the literature is that the trunk tilts away from the side involved in the unilateral sacroiliac strain. Goldthwait and Osgood,[35] Pitkin,[36] and Bucholz[37] have verified this. Their material was not confined to traumatic lesions, and in the early case presenting definite signs of sacroiliac soreness soon after injury I find the deviation homolateral in almost 100 per cent of cases. Later the inclination may change, or the curious phenomenon of alternating sciatic scoliosis may develop.[38] A peculiarity of these latter cases is that they have less pain in a posture of sciatic scoliosis toward either side than with the spine perfectly erect.[39] Pitkin[36] has recently been able to find only twenty-two cases described in the literature, but actually this condition is not so rare as these figures would indicate, as probably only the most spectacular cases have been reported. The same author found this phenomenon in 13.4 per cent at some time during the treatment of 506 cases of low back pain. The simplest explanation of these alternations is that there is a conflict between the tendency of the spine to deviate away from joint pathology in order to enlarge the intervertebral foramen or relieve pressure on the apophyseal or sacroiliac joints, on the one hand, and on the other a tendency for a reflex spasm of the quadratus and long muscles of the back to occur on the side of the lesion in an attempt to immobilize the painful joint.

Since the discovery of painful lesions due to posterior disc prolapse the suggestion has been made that a fair percentage of these alternating scolioses may be the result of an attempt to prevent pressure of this prolapse on the cauda by a deviation of the spine in either direction from the midline.

The Sciatic Syndrome. Most cases with complaints of sciatic symptoms in connection with low back pain, with or without an accident history, are not actually traumatic, although unquestionably a fracture of an articular facet, contusion of the sciatic trunk, or direct violence on the buttocks, a dislocation of the hip, or a forcible abduction of the thigh (as in doing the "split"), may injure the trunk or the lumbosacral plexus.[40]

After various types of sacralization were described by roentgenologists, Bertolotti[41] and others ascribed sciatica to this defect, but more recent studies by Moore[42] do not convince me that this anomaly is more frequent in patients with sciatica than in

others. Spondylolisthesis or lumbosacral arthritis, aggravated by trauma, and prolapse of the disc compressing the cauda are unquestionably occasional etiologic factors in the sciatic syndrome. Inflammatory changes in the piriformis muscle, probably secondary to sacroiliac pathology, have been reported by Freiberg and others[43,44] as occasionally compressing the nerve trunks.

Physical Signs in Sciatica. On inspection the patient may be seen to stand habitually with the hip of the affected side slightly flexed. He will have difficulty in walking up stairs when he bears weight on this leg. The gluteal fold is diminished. Sciatic scoliosis is common and the deviation is expected toward the opposite side. The patient avoids sitting squarely upon the buttocks. On bending forward in the sitting position the body tends to deviate toward the normal side if the sciatica is of root or funicular origin, and toward the affected side in some cases of injury of the nerve trunk. The patient may be unable to stand on tiptoes because of muscular weakness in the sciatic distribution below the knee.[45]

Palpation, besides revealing tenderness in the sciatic notch, may elicit other areas of tenderness in the sacral triangle or at the lumbosacral junction, depending on the location of the lesion. Rose's sign may be demonstrated in the radicular type. Tenderness extending down the course of the nerve and diminution in the consistency of the gluteal muscles or those of the calf may be found on palpation. Changes in sensation are typically over the lateral aspect of the calf and foot and paresthesia here may persist for years after the pain has disappeared.[46] The knee jerks are not diminished unless other roots than the fifth lumbar are affected. There has been considerable discussion regarding the interpretation of the straight leg-raising sign in sciatica. This sign is likely to be negative if the lesion is at the nerve root or funiculus, as for instance, in cases of pressure from a prolapsed disc or funiculus compression from intervertebral foramen changes, until a high degree of flexion of the leg is accomplished and is likely to be positive soon after flexion of the hip begins in lumbar plexus lesions and sciatic trunk contusions. This sign then has a quantitative value. It gives us some indication of the location of the painful lesion. The sign is evidently not pathognomonic for all types of sciatica.

At an early stage of sciatica or mild irritation of the roots or trunk, of whatever origin, most of these signs are not clear-cut or are entirely wanting.

Sacroiliac Injuries. In the decade following the publication of Goldthwait's work on subluxations and strains of the sacroiliac joints most low back injuries were accepted as being of sacroiliac origin. The discovery of much hitherto undescribed pathology by recently developed *x*-ray technique has caused a shift of opinion away from the diagnosis of sacroiliac lesions as a common cause of disability after accidents. Smith-Petersen's analysis of the distinction in signs produced by lumbosacral and sacroiliac pathology have made the diagnosis of lumbosacral injury much more common than formerly. Wilson and Danforth[47] in a careful anatomic and clinical study have convinced most men interested in low back injuries that radiation of pain through the sciatic distribution is produced by lumbosacral rather than sacroiliac pathology, and operative procedure for liberating the pressure on the fourth and fifth nerve roots by facetectomy, as recommended by Ghormley, has demonstrated that the lesion is commonly at this site or due to root or cauda pressure by a prolapsed disc.

In the ordinary case presenting classical findings of sacroiliac injuries neither *x*-ray nor clinical examination reveals actual subluxation of the joint. The pathology of these lesions, the symptoms of which are commonly relieved by manipulation of the joint, is not easily explainable. It seems to me likely that the lesion is commonly a sprain of the posterior sacroiliac ligaments and is similar to periarticular

sprains elsewhere, but until more is known regarding the production of pain by ligamentous injuries and capability of ligaments to develop spasm and contracture as muscles do, a reasonable explanation of why a single manipulation may produce a magical cure is difficult.

In severe soreness about a sacroiliac joint the gait is almost pathognomonic. The hip is not completely extended and the patient drags the foot. It is difficult to bear weight on the affected side in going up and, more especially, in coming down stairs. In sitting the weight is borne on the opposite ischial tuberosity. In both the standing and sitting positions there is commonly, in the more severe lesions, lumbar kyphosis, or a straight back as compared with the exaggerated lordosis common in lumbosacral strain. Sciatic scoliosis is not so prominent a feature as in lumbosacral strains. In the supine position, during straight leg lifting, a rotary strain can be placed on the sacroiliac joint of the affected side, reproducing the torsion that is caused by a patient falling and landing on his feet with the back straight. Further elevation of the leg transmits torsion to the opposite side, and when the patient is examined prone a rotation of the ilium on the sacrum in the opposite direction can be produced, so that if we know the position in which the patient sustained the injury it is possible to reproduce the same torsion on either sacroiliac. Such stress should exaggerate the pain. Unless the pathology is well advanced compressing the wings of the ilia together or separating them will not reproduce the pain on the affected side.

If any of the tests directed toward producing torsion on the sacroiliac joint is positive, any other explanation for the basis of pain except a sprain of the sacroiliac joint is difficult. Baer's sacroiliac point of tenderness in the abdomen is confirmatory if present.

It is notable that even extensive inflammatory disease of the joint may not yield the so-called sacroiliac signs. Thompson[48] reports a large series of cases of sacroiliac tuberculosis, in which only half presented diagnostic signs.

The Lumbosacral Group of Symptoms. In unilateral lumbosacral soreness the pain and tenderness are quite definitely localized between the fifth lumbar vertebra and the ilium. Even when there is extensive radiation of the pain in the sciatic distribution or over the gluteals the patient is likely to stand with a flat back or in lordosis and not with the lumbar kyphosis seen in a sacroiliac case. The sciatic scoliosis which is commonly seen presents a deviation principally confined to the lumbosacral region and not the gentle curve involving all the lumbar segments frequently seen in the sacroiliac case. This latter is also often accompanied by some rotation of the pelvis, not seen in lumbosacral lesions with sciatic scoliosis. In lumbosacral soreness uncomplicated by sciatic radiation the weight may be borne equally on the two sides in sitting and the lordosis of standing may be maintained in the sitting position. The amount of anterior flexion possible without pain varies little in the sitting as compared with the standing position, another distinction from sacroiliac soreness. On rising to the standing position after sitting, the patient may hold his back vertical and rigid. An attempt to stoop forward is confined to the upper part of the back and upper lumbar segments. The lumbosacral junction may be entirely immobile, or move only at the completion of flexion and at the end of the return to the upright position, causing a loss of rhythm. While climbing onto the examining table the weight is borne on the good side. In the supine position the leg raising is borne without pain until a considerable arc of motion has been attained and the stress is transmitted to the lumbosacral region. There may be only a slight difference between the range of painless leg raising on the normal as compared with the affected side, which is unlike the findings of sacroiliac strain. If both legs are raised simultaneously the pain is felt earlier than if they

are elevated separately. Confining the pelvis to the table during leg raising renders a greater arc of painless motion possible than if it is not so anchored.

In the side-lying position Gaenslen's test is negative unless very forcible extension of the hip is performed. If the patient suddenly releases the locked leg during this test pain ensues.

Data obtained on rotations of the trunk in the sitting or in the side-lying position are difficult to analyze and depends on the amount of sciatic scoliosis and attendant muscle spasm.

In general rotation of the trunk on the hips away from the painful side elicits pain at a smaller arc of motion than in the opposite direction.

Lateral motions are allowed through a greater range in the supine than in the sitting or standing position, and lateral manipulation away from the affected side is more painful than the opposite and painful in proportion to the acuity of the accompanying muscle spasm.

Lumbosacral lesions are the commonest type complicated by sciatic radiation and if the sciatic pain is of radicular or funicular origin the typical lumbosacral findings are obscured by signs of nerve irritation.

A posterior prolapse of the disc gives lumbosacral signs, much like those present in spondylolisthesis and lumbosacral arthritis. Percussion of the head or shoulders in the sitting position may cause exquisite localization of pain in any of these conditions. Rotation of the spine in extension is more poorly borne than with the spine in flexion. A unilateral sacralization of the fifth lumbar vertebra produces a painless sciatic scoliosis without muscle spasm unless there is an inflammation of the neoarthrosis of the deformed side.

Lumbar Arthritis and Lumbago. An unconvincing history of accident is generally given in cases of arthritis of the apophyseal joints of the lumbar spine. Pain is likely to begin suddenly while stooping or working in some disadvantageous position, the typical pain of lumbago. Certain positions are avoided, as for instance, leaning back in a chair while sitting, unless the spine is supported. The pain typically undergoes intermissions and acute exacerbations, becoming violent again after some unguarded motion. Examination not only reveals lumbar joint soreness but generally a superimposed myositis of the long muscles of the back. Spasm and increased consistency may be more pronounced on one side but is generally present to some extent in the muscle masses of both sides. In a localized arthritis there may be a lag in motion at one joint level if general muscle spasm is not too great to obscure this. Percussion of the head and shoulders causes a complaint of generalized lumbar pain or of localized pain if the joint lesion is localized. On stooping forward there is often no motion except at the hips and in the dorsal region. The leg-raising test can be performed in the supine position through a greater arc than in a case of pathology involving any joint adjacent to the sacrum. If both legs are raised there may not be much pain until the pelvis leaves the table.

In the prone position the head and shoulders can be elevated, producing a strain which travels down the spine with the amplification of the arc of motion until the stretching of painful spinal joints causes the patient to refuse further motion. Localized soreness on palpation may exist close to the spinous processes or in the lateral muscle masses. In a localized lesion percussion of the spinous processes is valuable in determining its location. Roentgenograms cannot be expected to demonstrate arthritis of the spinal joints until long after the fibrosis has rendered the spine almost perfectly immobile.[49,50]

Posterior Disc Prolapse Symptom Findings. "There are no physical, neurological or orthopedic signs which are found alone in cases of protrusion of intervertebral discs. All the known signs may also be found in other conditions."[51] Nevertheless, symptom signs may be so characteristic that a presumptive diagnosis can be made

before lipiodol roentgenographic studies are undertaken.[52]

Most of the physical signs are mentioned above and they are often unilateral. One expects to find the symptom complex of lumbosacral pathology, or "facet syndrome" with a sciatic scoliosis, generally deviating away from the side of the lesion. Commonly these signs are associated with those of sciatica of root origin.

Barr[52] analyzed the signs in 100 cases where posterior disc prolapse of the lumbar region was verified by operation and found lumbosacral pain in 70 per cent, sciatic radiation on the posterior thigh in 95 to 100 per cent, in the posterolateral aspect of the calf in 90 per cent, and over the lateral border of the foot in 5 per cent. Sciatic scoliosis was present in 60 per cent, and occasionally this was alternating.

The sitting and standing posture differs from that of spondylolisthesis and lumbosacral arthritis since the normal lordosis of the lumbar spine is generally lost and may be replaced by a fixed lumbar kyphosis. Barr found this condition in 90 per cent of his cases.

Rotation and lateral motion of the hyperextended spine in the sitting position are more painful than if the spine is straight or slightly flexed forward. This is explained by the tendency for the disc hernia to be reduced and thus cease to compress the cauda or nerve roots during flexion of the spine as demonstrated by Love and Walsh[51] and Schachtaschneider[53] in roentgenologic studies.

The spinal fluid findings have been mentioned above. The roentgenologic examination for disc herniation after lipiodol injection is described in detail by Hampton and Robinson.[54]

ADEQUATE ROENTGENOGRAPHY OF THE LOWER SPINE

If any case of disability following injury of the low back persists for more than a few weeks it can be expected to present on competent roentgenographic study positive findings suggesting the origin of the pathology, unless, of course, a trivial injury is overshadowed by inflammatory changes of non-traumatic origin in the muscles, ligaments or joints. The stimulus for much of the research on disc herniation, collapse of intervertebral spaces and changes about intervertebral foramina has been largely initiated by recent advances in *x*-ray technique. Adequate anterior posterior and lateral views are imperative in all *x*-ray examination of the low back. In addition to this, William's[55] method of visualizing the lumbosacral intervertebral space by taking the anteroposterior view of the patient supine with the hips partly flexed and a slight upward inclination of the orthogonal ray is a notable contribution.

Chamberlain[56] has studied sacroiliac mobility by taking posterior anterior views of the patient standing first on one leg and then on the other and measuring the amount of change in the relation of the two sides of the symphysis to each other. Evidence of increased mobility of a sacroiliac joint too small to be visualized in the roentgenograms of the sacroiliacs themselves is magnified by a shifting of position of the symphysis. He records a shifting of 0.5 mm. between the two pubes as normal in the male, while a little more motion can be found in the symptom-free female. A change in position of 2 mm. or more occurs only in the presence of sacroiliac symptoms. I have been able to satisfy myself of the absence of sacroiliac motion in back cases by this method.

In the anteroposterior view one obtains a fair visualization of some of the apophyseal joints of the first to fourth lumbar segments. Lange[57] finds that about 7 per cent of the lumbar joints are visualized in this position.

The lumbosacral apophyseal joints are only adequately visualized by oblique views. No routine roentgenographic study of the low back is complete without these. To obtain suitable oblique visualizations the body of the patient is rotated backward from the lateral position 45 degrees on the table. The lumbosacral articulation nearest

the table is visualized in this position. Both obliques must be taken. In the patient with scoliosis, however, some experimentation is necessary, with variations from the lateral position a few degrees backward or forward until these small joints are visualized.

Little was known concerning the pathology of the apophyseal joints and the fifth lumbar intervertebral foramen before the development of the technique for obtaining oblique lumbar views by Dittmar,[58] Ghormley and Kirklin,[59] Jordan[60] and Lange.[57]

CONCLUSION

The combinations of signs found in routine examination of lower back injuries are characteristic for the various lesions. The roentgenologic findings correspond to the physical signs, although no one sign is pathognomonic for any type of injury.

The protective muscle spasm and subsequent contracture affects definite muscle groups based on physiologic principles.

No special test for malingering is necessary, as no patient on repeated examination can maintain the limitations of motion and definitive signs of the various injuries consistently, or simulate the changes in function which correspond to the pathology found on physical and roentgenologic examination.

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THE EFFECT OF TRAUMA ON THE MECHANICALLY WEAK BACK

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THIS discussion will be limited to patients who have congenital or developmental variations, abnormalities or deformities of the lumbar and sacral regions of the spine.

In searching for the cause of pain and disability in the spine one cannot help being impressed by the large number of variations in the lower lumbar vertebrae and the first two segments of the sacrum. The fifth lumbar is subject to more variations than all the other vertebrae combined. Although these variations are congenital, they rarely produce painful symptoms during childhood or adolescence. This is probably accounted for by the fact that ossification and the normal restraining influence of ligaments has not been fully developed until adult life.

In comparing the skeletons of the biped and the quadruped the greatest difference is seen in the very acute bend which the biped lumbar spine makes to reach the perpendicular plane. Practically all of the change takes place in the last two lumbar vertebrae and the first segment of the sacrum. The wedged-shaped body of the fifth lumbar may be compared to the keystone of an arch, except that it is reversed; instead of being in a position to resist downward thrust, it is subject to shearing strain at its upper margin, and, to a greater extent at its inferior margin where it articulates with the sacrum.

This explains some of the anterior slipping or dislocations of the fourth lumbar on the fifth, and the much more frequent anterior displacement of the fifth on the sacrum.

If, in addition to the poor mechanics stated above, there should be a failure of fusion between the bodies and the pedicles of the lower lumbar vertebra, marked anterior dislocation of the fourth or fifth lumbar vertebra can occur. Many of these cases of anterior dislocations or spondylolisthesis have been reported. In a few rare instances the fifth lumbar has been known to slip entirely off the sacrum and dislocate into the pelvic cavity causing marked deformity and severe disability.

The treatment of spondylolisthesis depends to a great extent on the degree of displacement. The mild cases can be benefited by palliative measures such as corsets, braces or casts. The more severe cases require bone graft procedures before relief can be secured. For the exceedingly severe types there is little hope of complete relief even from bone grafts.

Other deformities of the bodies of the lumbar vertebrae may occur, such as, bipartate or hemivertebra, irregularly shaped vertebra or wedged-shaped from side to side. The treatment depends on the mechanics involved.

Failure of development of the laminae on one or both sides may be of little or no consequence.

The failure of development of several laminae may be accompanied by a large opening in the posterior aspect of the spinal column. Spina bifida may occur with or without meningocele. Small defects can be covered over by grafts extending from above down across and to the healthy bone below, if the symptoms in the area justify fixation.

Anomalies of the spinous processes are very common and usually give little or no trouble. There is occasionally seen in cases of increased lordosis, the impingement of one spinous process against the other with painful symptoms. Excising

the contacting bone until a gap is created will relieve the pain.

The transverse processes show greater variations than any other portion of the vertebra. The following are the most frequently noted variations:

1. One or both transverse processes may show increased length without symptoms. This condition is especially prevalent on the fourth and fifth lumbar vertebrae. However, it calls for no treatment.

2. The transverse process may extend straight outward to contact the rim of the ilium on bending to that side. The condition rarely calls for operative intervention.

3. The transverse process may extend outward and contact the ilium, forming a joint at the point of contact. This condition can occur unilaterally or bilaterally, and tends to limit the motion of the lower lumbar region, especially if bilateral. However, disability is more likely to occur when the contact is unilateral. In the latter instance, bending forward tends to rotate the fifth lumbar, as it is anchored on one side only. The abnormal motion is liable to cause painful symptoms following mild injury, or a seat of secondary infection may develop there from focal infections elsewhere in the body. The treatment consists either in excision of the projecting transverse process, or in fusing the fifth lumbar to the sacrum to stop all motion in the lumbosacral articulation, which is the seat of the pain. We prefer the latter operation.

4. This is the bat-wing type of enlargement of both the transverse processes, in which the processes extend downward and fuse with the sacrum by direct bone contact or by a joint. This is called "sacralized transverse process of the fifth lumbar." The condition may occur bilaterally without symptoms, but when unilateral, it may cause rotary torque and give rise to symptoms. Fusing the fifth to the sacrum usually gives entire relief from pain and disability.

5. The bat-wing type transverse process may extend downward and outward to form a joint with the ilium on either side, in continuation of the sacroiliac joint. The bilateral type very rarely gives symptoms, while the unilateral is very frequently the cause of rotary torque at the lumbosacral articulation with accompanying painful symptoms.

The treatment is to graft the fifth lumbar to the sacrum. If one cares to be more radical, he may fuse the sacroiliac joint on the side of the abnormal articulation. Curetting the articular facets between the fifth lumbar vertebra and the sacrum, reënforced by a generous supply of small chip grafts across the laminae, is entirely adequate.

Sacroiliac joints have been blamed for most low back pains in years past, and only in the last few years have they been exonerated. This blame on the sacroiliac has been due to the fact that the pain is referred to the region of the sacroiliac, even when the pathology is in the midline at the lumbosacral articulation. The pain may be constantly on only one side, but occasionally it alternates or is referred to both sacroiliac joints at the same time. The author has fused quite a few sacroiliac joints, using the technique of Smith-Peterson. Very few of these patients received permanent relief of pain in the lower back. Some returned with symptoms referable to the region of the opposite sacroiliac. In several instances the opposite sacroiliac was fused. In one instance, after fusing both sacroiliacs without relief, the lumbosacral was fused with complete recovery.

Under the influence of workers at the New York Orthopedic Hospital, more attention has been directed to the lumbosacral articulation. For a period we would fuse the lumbosacral and the sacroiliac on the side of the pain. These measures gave very satisfactory results with no tendency toward recurrences on the opposite side. Gradually, we were convinced that the pathology was in the lumbosacral and have, for several years, practiced fusing the fifth lumbar to the sacrum.

We have had entire relief of symptoms in about 80 per cent of those operated upon, and marked improvement in the others.

Fig. 1. An example of an exaggerated lumbosacral angle with horizontal sacrum.

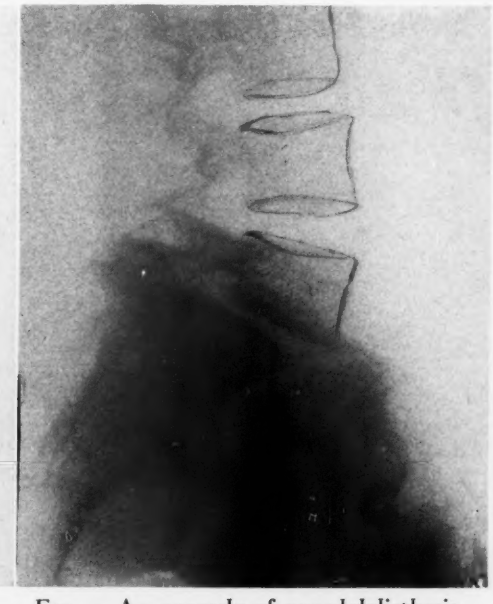

Fig. 2. An example of spondylolisthesis with failure of fusion of the pedicles.

In no case has the patient failed to receive some benefit. Sciatica has been a prominent symptom of several patients operated upon for pain in the lower lumbar region. In view of the present enlightenment of slipped intervertebral discs, we feel sure that some of the cases operated upon had pressure from displaced discs. However, these sciatica cases have responded almost as well as the other cases of bone grafting at the lumbosacral articulation.

We feel that in every instance of pain in the back, the slipped discs must be considered. If there is clinical evidence to support the suspicion of a slipped disc, the patient should have a thorough neurologic examination and protein estimation on the spinal fluid. Lipiodol injection should be used when indicated, but not used indiscriminately. If the fluoroscopic examination and roentgenograms show the disc protruding into the spinal canal then the removal of the cartilage should be considered if the symptoms justify.

The articular facets of the upper and midlumbar region, face internal-external in direction in the vast majority of individuals. On viewing the lumbar spine on an anteroposterior film, the joints appear as perpendicular planes and stand out quite clearly. Occasionally these internal-external type articulations continue down to, and include, the lumbosacral articulation. The internal-external is probably the most satisfactory type for function, and in our practice seldom causes trouble. The facets in the last few lumbar vertebrae may face anterior-posterior or obliquely on either side and still be excellent articulations so long as they are alike on the two sides. The asymmetrical type, wherein the articulations on one side may be internal-external, and those on the other anterior-posterior, are generally unsatisfactory, for while one joint moves normally the other is being moved at a right angle to its fellow on the opposite side. Mechanically, this is no better than moving the elbow or the knee laterally instead of forward and backward as nature intended. Although we often see articular facets at right angles on the two sides, it is more frequently seen that the variation is less than a right angle.

The asymmetrical type articulation is seen in such a large percentage of the

patients complaining of pain in the lumbosacral region, that one cannot help but feel that these joints are very easily

Fig. 3. A unilateral sacralized transverse process of the fifth lumbar vertebra.

Fig. 4. An example of asymmetrical articular facets of the fourth lumbar vertebra. One internal-external and the other anterior-posterior.

sprained in bending or twisting motions. They are also frequently seen in patients with pain in this region who have become disabled without history of injury. This type of injury usually is of short duration if treated by proper rest in bed, adhesive strapping, hot applications, diathermy, or an electric pad kept to the inflamed area. Alkaline salts, bulk forming or mild laxatives of non-irritating type (such as mineral oil or milk of magnesia), low carbohydrate diet, vegetables (especially the leafy and uncooked vegetables), fruits and fruit drinks with large quantities of water should be given.

Disability varies from three or four days to two or three weeks in the average case. Recurrences are likely to take place if any focal infection or acute infection develops, or if the patient allows his intestinal tract to become sluggish. In searching for focal infections, the teeth and tonsils are frequently blamed, but far too often the search is not sufficiently thorough. If relief or response is not secured, the gall-bladder, appendix and entire intestinal tract should be thoroughly *x*-rayed and otherwise studied. Also serologic and other laboratory tests should be carried out as indicated.

Repeated urinalysis will show the presence of high total acidity in the heavy carbohydrate eater, while heavy indican is likely to be present in the patient with cecal stasis or habitual constipation. If these attacks are infrequent and last for short periods only, the condition should be handled by removing focal infections and correcting the diet. If, after these measures have been taken, the attacks are severe or last for long periods, the patient should have an ankylosing operation on the joints involved. This does away with the painful motion, relieving muscle spasm, deformity, and disability. This type of patient, especially if his first attack occurs while performing some duty of his employment, is prone to blame the disability on a sprain or torn ligament.

If he happens to be employed under regulated compensation, he is alert to protect his rights and becomes a poor patient, especially when the surgeon begins to examine him generally and search for focal infections. He is willing to have local treatment to his back, but should one insinuate that the pus squeezed out of his tonsils, or pus expressed from the gums or the four plus indican in his urine, could have had anything to do with his "sprained back," he becomes suspicious. He stamps you as a "company" or "in-

surance" doctor and he not only distrusts you, but will probably carry out the treatment in a half-hearted manner. He may dismiss you in favor of "his family" doctor. If the patient does not promptly get well he is likely to believe that he is not being treated fairly, and that the company is trying to get out from under the responsibility of what he thinks is a real injury to his spine. On account of the ignorance concerning spinal injuries, more especially among the ignorant, the patient may conclude that he has a permanent disability. He naturally resists any effort to belittle the accident or blame a focus of infection as having anything to do with it, as he feels that this jeopardizes his chance of getting compensation, or, in case of permanent disability, any chance of satisfactory recovery.

He is not altogether to blame for his ignorance and his natural tendency to protect his rights. Very few back cases start off as malingerers, but many of them through ignorance, fear, or distrust of the employer, refuse to admit improvement until they get what they consider a satisfactory settlement of their claims. Due to the compensation aspect it is very difficult to know when ankylosing operations are indicated.

The muscle spasm and the lumbar scoliosis with tilting of the pelvis are our best indications that irritation is still present and that the disability still persists. In a large proportion of these cases, we feel hesitant to ankylose one or more vertebrae to the sacrum in compensation cases, although we would not hesitate in the case of a private patient. The compensation patient with a well fused (ankylosed) joint and all symptoms of low back irritation gone and apparently well, will in some instances receive as much compensation before an industrial board for the scar on his back as he would have received had he gone before the commission with an untreated sprained back. Therefore, insurance companies are justified in believing it better not to incur the additional expense of surgery, hospital and other necessary charges, until the industrial boards become more liberal toward the companies that are willing to give the patient the additional treatment. The public needs more enlightenment on the many disabilities that can occur in the spine, their causes and corrective measures. Then and then only will the laity dismiss the idea that all spine injuries cause permanent disability.

CONCLUSION

In conclusion, it is our experience that the anomalies mentioned above are a definite source of pain in the low back when aggravated by injuries to the back, or when affected by toxemias. It has been observed that in the presence of such anomalies, disability can result without injury. The presence of such symptoms as muscle spasm, scoliosis, lordosis or tilted pelvis, although definite evidence of irritation, do not necessarily prove an injury has existed.

DIAGNOSIS AND TREATMENT OF ACUTE AND CHRONIC BACK PAIN DUE TO TRAUMA

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IN presenting this paper, I should like it thoroughly understood that it deals only with the traumatic phase. I was fortunate enough to be associated with Sir Robert Jones during the World War, and through his kindly aid, learned his methods of handling these injuries both in the diagnostic problems they presented and from a therapeutic standpoint. Since the War I have applied Jones' teachings and have obtained successful results; these serve as a basis for this report.

I am adhering rigidly to traumatic backs. In none of these cases have I been able to find the so-called sacroiliac strain, subluxation, or luxation. Frankly, I do not think there is any such entity, an opinion which I base on the fact that the pelvis is so well mortised that a subluxation or luxation cannot be produced without fracture; furthermore, the photographs which accompany this paper illustrate that a back wrenching calls for every bit of strength possible. Yet, I have never been able to produce under the most strenuous circumstances, a post-manipulative luxation or subluxation.

I realize that this paper will call for a lot of criticism, particularly among orthopedists, but I hope they will keep in mind that I am only referring to traumatic backs. It is essential to examine each case carefully; a hasty diagnosis will unquestionably yield a poor result.

Since opinion differs so widely as to the origin and causation of back pain, the methods of treatment have naturally been numerous and varied and have produced inconsistent results. On the whole, a definite program of treatment which can be used by the general practitioner in these cases has been wanting. Since it is he, rather than the orthopedic surgeon, who usually first sees these acute back injuries, it is important that a rational plan of therapy be carried out from the beginning.

In the cases presented herein those back injuries involving fracture to the spine, exhibiting bone or cartilage pathology or showing the usual arthritic changes found in the middle or old age groups, have purposely been omitted since they are definite, recognizable entities in which the causation and treatment of the back pain are fairly well established.

In consideration of the treatment of low back pain arising from trauma, the mechanism of its production is important and it is here that so much controversy has arisen. The anatomic structure of the back has been elaborately reviewed along with various theories and clinical studies of the mechanics of production of pain.

Owing to the relatively small intervertebral spaces in the dorsal segment compared to the lumbar region, and also because of the vertical direction of the articular processes and overlapping of the spinous processes, motion in the thoracic spine is greatly limited. Flexion, extension and lateral motion are greatest at the level of the second, third, and fourth lumbar vertebrae.

The principal extensor muscles acting on the lumbar and thoracic regions are the erector spinae on each side and its principal components, of which the longissimus dorsi is most important. The erector spinae is attached to the back of the crest of the ilium, posterior sacroiliac ligament, back of the sacrum and the spines of the lumbar vertebrae. It is placed at the root of the spine dorsally on each side. The spinalis dorsi is attached entirely along

the spinous processes arising from the spines of the lumbar and lower two dorsal vertebrae and inserted into the spines of the upper dorsal vertebrae. The longissimus has an attachment to the transverse process of the lumbar vertebrae and a double set of insertions to the transverse process of the thoracic vertebrae and also into the back of all the ribs. The intertransversales muscles extend between adjacent transverse processes of the vertebrae and occur principally in the lumbar segment where they are well developed and aid in lateral movements of this part. The erector spinae muscles act as extensors and maintain the erect attitude of the spine preventing forward falling of the trunk.

The rotatores spinae occur in the thoracic spine and extend on each side between the transverse process of one vertebra and lamina of the vertebra above. They rotate the dorsal vertebrae and turn the trunk to the opposite side. The multifidus spinae occur in the cervical, dorsal, lumbar and sacral regions. In the lumbar segment they arise from the articular processes and in the thoracic from the transverse processes and are inserted into the spinous processes of the vertebrae above, some of the fibers passing between adjacent vertebrae, others to the second, third and fourth vertebrae above. In the thoracic region the left and the right groups act together and help to prevent the spine from being bent forward. When the muscles of one side are contracted they assist the multifidus spinae in producing rotation to the opposite side. In completion, the anterior flexion of the spine is produced by the quadratus lumborum assisted by the psoas muscles acting as antagonists to the extensor group. Their rôle in the mechanism of back sprains is less important.

In the majority of instances traumatization is confined to the soft tissues and supporting structures of the back and arises from a sudden increase in stress and strain upon them. Sudden wrenching causes tearing at the bony insertion of the muscle tendon or stretching and tearing of the muscle belly itself, resulting in diffuse injury to the elastic tissue about the tendon and to each muscle fiber or bands of muscle fibers; hence, an adaptive shortening of normal structures may arise from decreased mobility and contracture of involved muscles. Any forced movement which ordinarily would be well within the normal anatomic limits would be restricted due to contracted tissues and would result in a sprain, producing pain on movement.

The majority of back injuries occur from lifting, twisting, and jumping or from direct blows. As a result, the erector spinae groups are the main structures involved. This is evident clinically by spasm and accurately relocalized tenderness over this area, particularly at the posterior sacrosciatic notch, usually unilateral and occasionally bilateral. Autero-flexion or lateral rotation of the trunk usually increases the discomfort and these motions are therefore limited.

In long-standing cases healing of torn, overly stretched muscle results in fibrosis and adhesive bands across the muscle bundle and fibers with resultant contraction and shortening. It is understood that associated muscle groups of the back may be similarly affected by the same type of trauma and give rise to parallel symptoms, though this is more uncommon. Referred pain from adhesions and fibrosis of these muscle structures and involvement of the sciatic nerve with the development of a traumatic sciatica is occasionally seen.

In the differential diagnosis of traumatic backs four general types are recognized:

I. True back injuries of acute or chronic variety.
II. Neurotic backs
III. Malingering backs
IV. Pathologic backs.

1. In the *true back injury* a definite history of trauma is obtainable. This was usually followed by immediate disability. Inspection reveals a change in postural position favoring the affected side. True limitation in trunk motion in all directions

is present because of definite spasm and rigidity of the musculature of the affected side. The exertion of digital pressure over the erector spinae muscles at the posterior sacrosciatic notch causes immediate expression of subjective pain which never varies; there is no confusion on the part of the patient as to the place involved.

X-ray showing both anterior-posterior and lateral views of the spinal segments involved is indicated in every case in which a fall or a blow to the back occurred. Different angular views are occasionally necessary to secure adequate bone detail and definitely ascertain whether injury to the osseous structures exists.

In chronic low back pain the patient gives a history of injury, followed by prolonged disability and often many types of unsuccessful treatment. The symptoms and signs are more or less constant pain on the affected side with postural contraction toward that side, inability to bend with comfort in all directions and definite muscle rigidity or protracted spasm and pain on pressure over the posterior sacrosciatic notch or the affected muscle group. In examining these cases a red skin pencil mark is made over the sites of pain and repeated palpation over these points yields definite relocalization of subjective pain. In some instances pain along the sciatic nerve of the affected side is described. Bilateral sciatica in uncommon.

II. Back pain is sometimes associated with a *traumatic neurosis* developing in certain types of individuals following a true back injury. Neurosis develops after extensive varied treatment has enlarged in the patient's mind the degree of injury. Suggestions of friends, emotional instability, domestic troubles, and related conditions are underlying causative factors in the development of this type.

The general appearance of the subject is typical. He appears worried, apprehensive of the examination and highly nervous, but he is open-minded and sincere in describing complaints. Stripped of their clothing for examination these patients are asthenic, perspire freely, and show increasing nervousness as the examination progresses. The back musculature is soft and underdeveloped and any digital pressure causes pain but usually on deep palpation. Relocalization of painful areas is not accurate; there is no limitation in bending in any direction and no muscle spasm or rigidity.

Winning the confidence of the patient is all important in these cases since successful treatment depends on his cooperation and belief in the surgeon's ability to help him. Thoroughness of examination and a sympathetic ear to complaints accomplishes a great deal. Deliberation in diagnosis, even to the extent of suspending an opinion for several days or weeks, is most impressive. Several examinations and oftentimes laboratory tests are necessary before confidence is established. Roentgenograms are always essential in this type of back pain to rule out possible bone pathology.

III. Usually the man who comes in with a cane or two canes and even crutches a long interval after an injury must be regarded with suspicion. He presents a belligerent air, is wary of every move you make, objects to being questioned, wants one or two friends in the examining room and refuses to give a clear concise story. Often he has worked only a short period for his employer or scarcity of work has caused him to seek disability benefits for a non-disabling injury. Unobserved inspection of this type of individual while he is putting on and taking off his clothing often gives an accurate impression of the extent of the back injury. The true back motion is disclosed in his ability to tie shoe laces, pull one trouser leg on and then the other either in a stooped or straightened position and by agility in sitting down or standing up.

The skin pencil test in these people will show anywhere from ten to fifteen painful spots which on repeated tests cannot be relocalized. Incidentally, examination before a mirror is to be avoided since the

examiner's movements can be too easily premeditated by the patient. Again, light digital pressure over the allegedly affected area, even though it is a mere stroking of the skin, causes flinching and much complaint of pain. No true muscle spasm, or spinal limitation can be found present although active trunk motion may be restricted through a range of 10 to 15 degrees. A revealing test which the writer has developed and calls the "football kick test" is used as a means of determining localization of back pain. The subject is asked to dropkick an imaginary football. The purpose of this is to put sudden strain upon the lower back. Kicking is done first with one, then with the other leg. Patients with true back pain manifest pain accurately over the affected area of the back but perform the test without hesitancy. The malingerer refuses to perform the test or so grossly exaggerates that feigning becomes obvious.

IV. Frequently people sustain back injuries which aggravate underlying pathology of the spinal column and although this report is not concerned with this type of back, the importance of the roentgenogram in differential diagnosis cannot be overemphasized.

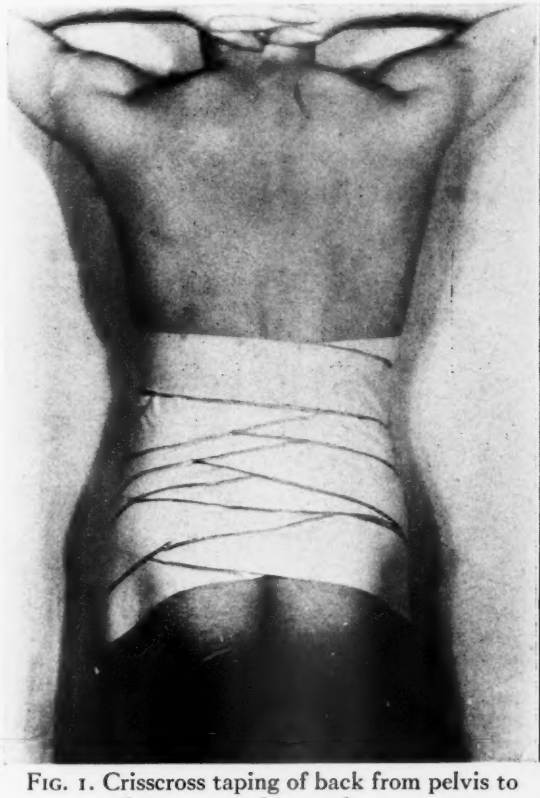

Fig. 1. Crisscross taping of back from pelvis to ribs to secure firm anchorage.

Fig. 2. Side view of back taping. Edges of tape have been lined to illustrate crisscross effect.

From this consideration a method of treatment suggests itself. In acute sprains the first objective is to put the injured tissues at rest and thereby relieve spasm and pain. Anodynes, of course, are often necessary adjuncts to promote symptomatic relief. Rest of the injured muscles and soft tissue structures is secured by adhesive immobilization of the back, usually preceded by infra-red heat or other forms of topically applied heat to produce relaxation. Three-inch strips of adjesive are used, so placed that an anchoring of the pelvis to the thoracic cage by crisscross taping is produced. The accompanying diagram (Figs. 1 and 2) serves to illustrate this point better.

After two to three days the adhesive is removed, infra-red heat again applied and Swedish massage of the back and

thigh muscles commenced. The purpose of the massaging is twofold. It serves to relax muscular spasm and to aid the

FIG. 3. Patient in deep ether anesthesia. Assistant fixes pelvis. Operator grasps subject about shoulders and wrenches trunk to right.

FIG. 4. Same procedure as in Figure 3, but to left.

dispersion of exudate within the soft tissue substance. Further adhesive immobilization is sometimes found necessary after heat and massage for the first ten days. Prolonged immobilization either with tape or rigid supports such as braces or casts is discouraged because it is found that unless early mobility is achieved, muscular wasting occurs, with resultant pain, stiffness and weakness that takes a long period of treatment to overcome. The foregoing measures are carried out until the patient obtains relief or until it is felt that clinical recovery is complete.

In considering chronic back pain the approach to the treatment assumes a different aspect. Unfortunately in traumatic work in which so many of the cases are industrial accidents and come under the category of compensation insurance, we are harassed by frequent instances of deliberate malingering to prolong disability payments. Hence, with individuals who come to our attention with histories of long-standing back pain or even in patients originally seen who had acute injuries but continue to complain of persistent pain after prolonged treatment, one must determine whether they are suffering from true chronic back pain, or have developed a traumatic neurosis responsible for their symptoms, or whether they are out and out fakirs.

Often in genuine chronic back pain a persistency of the pain may be associated with absorption from foci of infection in some other portions of the body such as abscessed teeth, infected tonsils, chronic prostatitis, etc. These, of course, must be ruled out or eliminated before further management can be considered. Postural defects are common, particularly in inherently weak backs in which muscular development is poor or lacking and the subject has a lordotic stance or contracture.

The operative program for chronic cases is confined to wrenching the back under deep anesthesia following the method used by Sir Robert Jones. Before any wrenching procedure, care is taken to make roent-

genograms of the spine to rule out bone pathology, fresh fractures or advanced arthritis. Patients are admitted to the hospital eighteen hours before and preoperatively are given morphine and atropine. The technique is described as follows and illustrated in the accompanying pictures. (Figs. 3 to 8.) With the subject in the third stage of surgical anesthesia the assistant fixes the pelvis to the table by pressure and body weight directed over the iliac crests. The operator then encircles his arms about the upper trunk and arms of the patient and with a forcible jerk bends the trunk laterally. The same maneuver is repeated from the opposite side. The third step is conducted by forcibly jack-knifing the subject over the operator's knee. This is then repeated with the patient in a supine position; in this instance merely allowing the trunk to hyperextend without added force applied is sufficient. The fifth and sixth steps consist of flexing the thighs on the abdomen and forcibly extending the legs.

Fig. 5. Operator places his own knee on table and, with subject in prone position, forcible jack-knifes trunk.

Fig. 6. Same as in Figure 5, except that patient is in supine position and trunk is allowed to hyperextend without added force.

The patient is returned to bed and is kept at complete bed-rest for ten days. No traction or other forms of treatment are given at this time. Often complete relief is immediately obtained; in others partial relief is secured and the program instituted after dismissal from the hospital consists of massage and heat, muscle reëducation and postural exercises. In the event the case is based on hysteria, the ether anesthesia and wrenching serve as a psychotherapeutic procedure. It has been found that suggestion of complete relief while the patient is being anesthetized has considerable bearing on the outcome since it fixes the thought in their minds immediately prior to unconsciousness.

The preceding methods of treatment have been used for several years. The following survey is based on approximately 400 cases treated since 1935. Arbitrarily, the causation of accident was classified under four general headings—(1) lifting, (2) falling or jumping, (3) striking (that

is, either by striking the back against some object or by an object falling upon the back), and (4) twisting. Of these, lifting was the most common cause and constituted 56 per cent of the cases. The usual stories of injury were similar to these—"placing a box of tea high on a shelf, felt a sudden catching pain in the back"; "moving heavy plank felt something pull loose in the back"; "lifting washtub of water, heard a snap in the back and was immediately disabled by sharp pain." Falls or jumping accounted for 17 per cent of the accidents and these were from heights varying from 1 to 30 feet. Blows to the back accounted for 16 per cent of injuries; lastly, 11 per cent of the accidents were caused by twisting.

The average age of these patients was 34 years, the youngest being 16 and the oldest 61. It was also noted that the younger individuals responded more quickly to treatment with considerably less disability while under medical care. The greatest majority were males, but this is natural since most of the cases were industrial accidents. The average disability was fourteen days with average recovery period of thirty days. The longest disability and recovery period was 120 days and the shortest, no disability and recovery in five days. In computing these last figures certain arbitrary opinions were necessary in some instances in determining disability and recovery periods. These included instances where recovery was judged complete in our opinion and the opinion of one or more medical referees to whom the cases were referred for examination. In many cases previous medical care had been given the injured, and in some cases they had failed to report the accident for several weeks, thereby delaying treatment.

In this series it was found necessary to do back wrenching in only nineteen cases. The results were more gratifying on private patients than on compensation cases.

Fig. 7. Flexion of thigh on trunk.

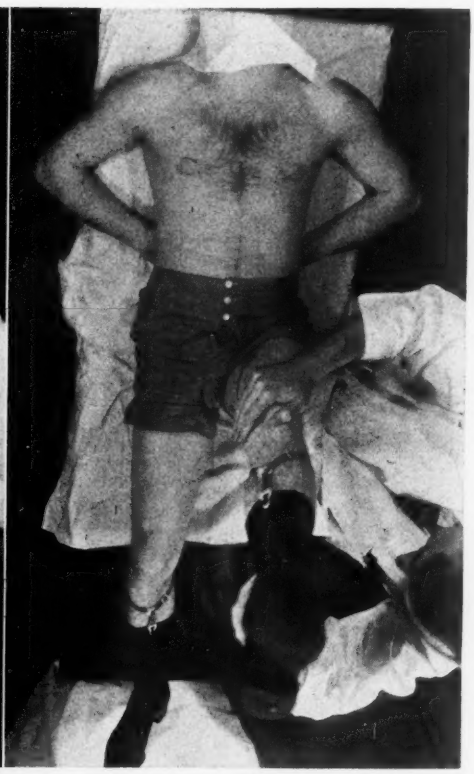

Fig. 8. Extension of leg after thigh has been flexed on trunk. Figures 7 and 8 represent maneuvers that are carried out on both lower extremities.

Other statistics in the total group showed that forty-nine failed to return for further treatment and follow-up checks on the greater majority of these indicated that they had recovered and did not feel the need of further treatment. Twenty-nine patients were ascertained to be malingerers, in whom response to treatment was unsatisfactory. Of these, six cases were heard and disposed of before the Commission. Ten cases were reëxamined from three months to one year following discharge from treatment and in each instance the objective findings were negative.

Anomalous and congenital changes of the spine as spina bifida occulta, rudimentary ribs and vertebral bodies, fifth lumbar sacralization, etc., were not uncommonly present. Roentgenograms were taken in seventy-five cases. Definite sciatica developed in nine patients. Back wrenching and sciatic stretching were performed in five patients with relief in four and no improvement in the other.

Of the nineteen cases wrenched, eleven admitted complete relief of their back pain. The remaining eight, although admitting partial or complete relief after the wrenching, later denied relief; of these, seven were compensation cases in which Commission action denied further benefits. The other was an instance of sciatica of long standing in a private patient who had received various treatment for several years before coming to our attention. Several cases of back-wrenching are briefly described here.

Case I. E. R. (personal), 42 years of age, developed shooting, cramp-like pains in the small of the back and down the back of the right thigh and leg in May 1937. Pain continued without relief until he was admitted to the hospital in July. Medication, consisting of salicylates, mud baths, diathermy, and salt baths, had been tried without effect.

The past history was essentially negative. All known foci of infection had been removed.

Examination revealed a sciatic neuritis with subjective pain in the right lumbosacral region of the back and pain radiating along the right sciatic nerve and posterior aspect of the leg. Some atrophy of disuse was present in the thigh and calf muscles of the extremity. Back wrenching and sciatic nerve stretching were done under general ether anesthesia. The following day the patient admitted complete relief of pain, and he was discharged from the hospital after a week.

A letter from the patient, received on March 8, 1938, stated that he was completely relieved from his back and leg pain, that he had been able to resume his regular work the day following his dismissal from the hospital, and that he has driven his car from 500 to 1,000 miles each week during the past year without discomfort.

Case II. R. L. (compensation—malingerer), on October 24, 1934, was lifting a gin-pole when he experienced a sharp pain in the back. He received treatment and was well until December 12, 1935 when he slipped from a flat car while working on a concrete mixer and fell 4 feet to the ground, landing on his back.

When referred to us on March 18, 1936, he complained of pain in the lower part of his back, inability to work, and no improvement in the condition from the date of injury. Past history was essentially negative. Examination objectively was entirely negative, but the patient alleged indefinite pain over the left and right sacroiliac regions and in the midline of the lumbar segment. All alleged symptoms appeared grossly exaggerated. *X*-ray studies of the lumbar sacral region were negative for old or recent fracture or any bone pathology and showed only a sacralization of the fifth lumbar transverse process on the right side. Our opinion was that the man either was suffering from a traumatic neurosis or was a malingerer.

A suggestion of operation to alleviate his back pain was accepted, however, and on March 25, 1936 back-wrenching was performed under general anesthesia. On March 31 he was discharged from the hospital. He remained under treatment of heat, massage and back muscle training exercises until discharge on May 28, 1936.

He continued to profess inability to work and the case was carried into litigation, at which time the Commission disallowed further benefits. Reëxamination on June 12, 1936 revealed the clinical picture unchanged and all objective findings and roentgenograms normal.

Case III. G. C. (personal), in August 1935, was kicked in the right hip by a horse. Soon after he developed pain in the lower right back and felt a "tightening" in back of the right thigh. This increased in severity until September 10, 1936. When first seen, he had developed a constant aching pain in the lower back and in the posterior aspect of the right thigh. *X*-ray of the dorsal and lumbar spine appeared negative. Diagnosis of an old traumatic myositis of the erector spinae musculature and lower back muscles was made.

Under general anesthesia the back was wrenched. The patient was hospitalized for only two days, and upon discharge was practically pain-free. No follow-up treatment was given and the patient has had no recurrence of his discomfort. When examined on March 2, 1937 he demonstrated full normal trunk motion without subjective discomfort.

Case IV. E. F. (compensation—neurosis), was loading an empty stretcher weighing about 75 pounds into an ambulance in January 1935. In lifting and twisting to the left he had a "stitch of pain" in the small of the back. He continued his work and an hour later while helping to lift a 160 pound man out of a bathtub felt another "stitch" in the back. Pain was so severe that he could not straighten up and had not been able to stand erect since. He was forced to bed for two weeks and then was hospitalized for thirty-four days. He returned home, but developed more severe back pain. He had not worked from January 7, 1935 to December of that year.

His complaints at time of examination on December 9, 1935 were constant low back pain with limited motion, pain in the thigh and leg and vomiting. Examination of the back was objectively negative and stereoscopic *x*-rays of the lumbosacral spine were negative. A diagnosis of a traumatic neurosis was made and back wrenching suggested to the patient as a means of cure for his back pain. This he accepted and operation was performed on December 10, 1935. Following three days of hospitalization he was given several treatments of heat and massage and muscle-training exercises and was discharged on December 16, 1935 completely relieved of his back pain and with normal back motion.

Case V. C. S. (compensation—malingerer), while lifting a 450 pound motor on January 7, 1935 with the aid of several others, had a sudden sharp pain in his back. He was assisted home and reported to the doctor three times a week for physiotherapy until May 1. Since he did not improve, he was sent to a hospital where teeth were removed and he received further physiotherapy to his back. He remained in the hospital till the end of June at which time he was discharged and a belt applied; this he has worn since with much relief of back pain but inability to work. He could walk slowly for only short distances and then with the aid of a cane.

Examination revealed definite muscle spasm of the right erector spinae musculature with accurate relocalization of pain in this area and restriction in trunk motion. *X*-rays of the lumbosacral spine were negative.

Back wrenching was proposed and accepted. This was done on January 26, 1936. On January 28, the patient admitted complete relief of back pain and desired to return to his home (Tennessee). Compensation was then cut off. Within a week he commenced having back pain again and professed total disability.

On August 6, 1936 he returned for reëxamination at which time objective findings were completely negative and opinion was that the man grossly exaggerated his symptoms. Further referee examinations at a later date disallowed further benefits and it was concluded that the man was able to work in spite of his protests.

CONCLUSIONS

It is hoped that this treatment of low back pain may be of some help to others and that it be given a fair trial before being unjustly criticized. Emphasis on radical procedures is a present-day trend in this ever-increasing problem, and it is hoped that this report will justify and encourage the use of conservative therapy.

I wish to thank my associates, Drs. H. P. Stimson, A. H. Mason, and C. C. Coleman, for aid in the preparation of this manuscript.

TRAUMA OF THE COCCYX AND COCCYGODYNIA*

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SINCE Simpson's application of the name coccygodynia to persistent pain in the neighborhood of the coccyx there has been much speculation as to cause and treatment of this often intractable condition. The extent to which trauma is the cause, the part played by neurosis, whether palliation or operation is to be the accepted line of treatment are all matters over which there has been more or less continuous disagreement.

By and large, few conclusions have been drawn as to etiology and there is still wide variation in opinion as to the relative merits of the therapeutic measures in general use.

The reason for these uncertainties lies in the absence of any uniformity in changes, physical or pathologic, that are demonstrable in this disease. Whether the trouble lies in the bone, its coverings, in the joint, in the nerves or the muscles in the vicinity has not been satisfactorily determined, so that whatever treatment is adopted is bound to be governed by the variety of empiricism that is used for similar unexplained pain elsewhere.

Anatomically, although the coccyx would appear to be particularly prone to injury, it actually enjoys a real measure of protection from the surrounding structures, more so in the male perhaps than in the female. Falls upon a plane surface, even though occurring in the sitting position, are far more likely to throw impact upon the ischial tuberosities or the dorsum of the sacrum than upon the coccyx, and in any case are bound to be cushioned to a certain extent by the gluteal muscles. Only angular objects, such as may enter the natal cleft, can be expected to inflict any appreciable amount of damage to the coccyx or to the tissues nearby.

There is no doubt that trauma plays a major part in the production of coccygodynia, but contrary to what is frequently assumed, major trauma such as fracture or dislocation is rarely encountered. Paradoxically, the degree of injury is in no way proportional to the persistence and intensity of the subsequent symptoms. In those few cases in which deformity can be positively ascribed to injury, there is often much less difficulty in management than where there has been but little obvious damage.

Physical examination most often reveals nothing of a very convincing character. Some have sought to emphasize the importance of such features as fixation, others of undue mobility of the coccyx. Further stress has been placed upon deformities, either in the form of angulations forwards at various levels or of deflections to one or other side. Roentgenologic confirmation has been presented to show that such deformities exist, but whether they are the consequence of injury is another matter entirely. In this respect Duncan[1] has made an interesting comparative *x*-ray study of 262 cases with painful coccyx and 200 without. The percentages of anterior angulation and lateral deviation showed little difference in each group. Similarly, in the plain digital examination of these patients there will be encountered as many fixed as excessively mobile coccyges.

The location of pain and tenderness is not by any means constant. Confined sometimes to the coccyx and its borders, or to the sacrococcygeal joint, it is as often located in the muscles to each side, which lends some support to the thesis of Thiele[7] that the pain is the consequence of spastic

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contraction of these muscles, akin to the pyriformis spasm of orthopedic interest.

As far as differential diagnosis is concerned, too much stress cannot be placed upon the necessity for thorough examination of all other structures possibly involved. Useful information may be obtained from neurologic investigation, particularly as regards organic disease. Sufferers from the rectal crisis of tabes dorsalis not infrequently consult the proctologist first and because of the apparent absence of accompanying physical signs the pain may be ascribed to coccygodynia. However, a relaxed sphincter will direct the attention to other neurologic phenomena. Lesions of the conus medullaris, often traumatic in origin, and occasionally mentioned in connection with coccygodynia, are more often productive of partial anesthesias and paralyses of saddle distribution.

Rectal lesions such as abscess, fissure, blind internal fistula, are generally easily recognizable, but it must be borne in mind that they may accompany an offending coccyx, or that coccygodynia may even be a sequel to operation for their relief.

In the treatment of coccygeal trauma and coccygodynia, both conservative and aggressive measures appear to play about an equal role. In recent injury rest, heat, sedation and attention to the bowels are called for as the severity of the condition demands, and the more distressing symptoms will in many cases subside. It is very questionable whether anything is achieved by manipulation of the coccyx or reposition of presumably detached fragments under anesthesia at this stage. Rarely such a course may appear to be indicated, but because of the inability to maintain alignment it would be just as rational in the circumstances to perform excision, which, of course, brings us back to the question of which deformities are really of traumatic origin.

For persistence of pain, one has the choice of a variety of therapeutic measures, singly or in combination. With the idea of affording protection to the irritated part, some use may be made of the padding provided by the soft parts of the nates. This may be accomplished by firm strapping or the wearing of a well-fitted girdle, as suggested by Key and Conwell.[6] Something similar may be achieved by instructing the patient at all times to approximate the buttocks before sitting.

Although it is often advised that sitting will be more comfortable upon upholstered furniture or upon a rubber ring, in reality less pressure is exerted on the coccygeal region if the patient sits erect on a hard wooden chair, the weight of the body being supported by the thighs and ischial tuberosities.

Massage and manipulation continue to play a deservedly prominent part in the treatment of this condition. To quote Ely,[2] "It consists of massage of the coccyx by means of the forefinger in the vagina and the thumb on the outside, holding the bone between them. The bone is moved backward and forward and the soft parts are moved about on the bone." Needless to say, the finger in the rectum serves just as satisfactorily. Duncan[1] emphasizes forcible extension of the coccyx at the sacrococcygeal joint as an essential part of the technique. Thiele,[7] who explains coccygodynia on the basis of levator, coccygeus and pyriformis spasm, requires that the finger be fully inserted within the anus and the massage applied by sweeping movements across the posterolateral aspect of the pelvic floor. Treatments of this type may be given for one or two minutes every day or every other day at first, the interval being increased in porportion to the degree of relief.

The injection of analgesic agents demands some consideration. Yeomans' technique consists in the repeated injection of 10 to 20 minims of 70 to 80 per cent grain alcohol into the point of maximum tenderness, determined by pressure upon the parts between the finger inside and the thumb outside the rectum. An interval of one week is allowed to elapse between each

injection and as many as ten have been given, although the average is four. There is considerable pain at the time of and for several minutes after this procedure and a dull ache persists for twenty-four hours. If success is to be expected by this method in any particular case, some relief is to be looked for after the first few injections. Other agents that have been recommended are quinine-urea hydrochloride, and procaine in varying strengths. Epidural injections of saline and procaine are mentioned only to be discouraged.

Operative treatment varies from the subcutaneous division of the structures attached to the coccyx, originally practiced and discarded by Simpson to removal of varying amounts of the coccyx and sacrum. The operation of excision presents no great difficulties and has been performed through longitudinal, U-shaped and transverse incisions. There is a remote danger of perforation of the rectum. Approximation of the detached muscles is called for, and with reasonable care infection can be avoided. Key and Conwell ascribe their unusual percentage of cures with excision to the removal of the tip of the sacrum and leveling off of the stump.

At what time during the course of this disease any of these therapeutic measures is to be adopted is a question not very easily answered. Some are inclined to continue palliation of one sort or another for six months or more. Others prescribe immediate excision as soon as the diagnosis is made to forestall the establishment of neuralgia, neuritis, or the particular intangible to which they attribute the subsequent pain. Not to be overlooked is the possibility of spontaneous cure in certain cases which may figure as evidence of the efficacy of some favored method of treatment.

TABLE I

	Method of Treatment	Number of Cases	Traced	Cured	Improved	Unimproved	Percentage Unimproved
Duncan	Conservative	248	54	52	..	2	3.7
	Excision	30	30	22	3	5	17.0
Hamsa	Conservative	24	24	18	..	6	25.0
	Excision	49	49	40	..	9	18.4
Thiele and others	Massage	80	80	48	27	5	6.3
Hobart	Massage	15	15	8	2	5	33.3
Key	Excision	15	14	12	2	0	0.0
Yeomans	Injection	24	24	16	7	1	4.2
Waters	Injection	53	49	41	6	2	4.1
This series	Excision	37	16	8	6	2	12.5

The results obtained by various observers and the methods used are given in the table below. Included is a small series of cases operated on at University Hospital since 1922. Of thirty-seven excisions, sixteen could be traced; eight were said to be cured, six were improved and two unimproved. It will be observed that there is in each series a certain number of intractable cases. What to do with them has not been determined and no solution is offered here. This much, however, is certain, that wherever there is a motive for the perpetu-

ation of symptoms, there will be considerable difficulty in handling the case. This applies particularly where there is a compensation factor concerned and much caution is to be exercised in these circumstances in recommending excision.

SUMMARY

Attention is directed to the necessity for care in the interpretation of physical signs and *x*-ray findings in cases of trauma of the coccyx and coccygodynia. Major trauma is uncommon. Because of the frequent failure of excision to effect a cure, conservative measures should be persisted with for longer periods than is generally the practice.

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The treatment of fractures of the bones of the leg requires frequent checking and control by *x*-ray examination. This is the only means of determining whether reduction has been obtained, whether bony alignment is being maintained afterward and whether the splinting methods are effective.

A PLEA FOR CONSERVATISM IN FUSING OPERATIONS ON THE SPINE AND PELVIS

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IN 1891 Hadra conceived and purposely executed an endeavor for the immobilization of the spine by wiring together the spinous processes. The wiring was done for fracture dislocations and later for tuberculosis of the vertebrae. Hadra thought up the idea independently and has been so credited. It has been my good fortune to have discussed with later associates and friends his persistence and the conclusion he was finally forced to accept in his efforts to immobilize the tuberculous spine. Thus the institution at which he practiced and with which I have been associated early was convinced of the conservatism of spinal fusion, and this has greatly influenced our methods.

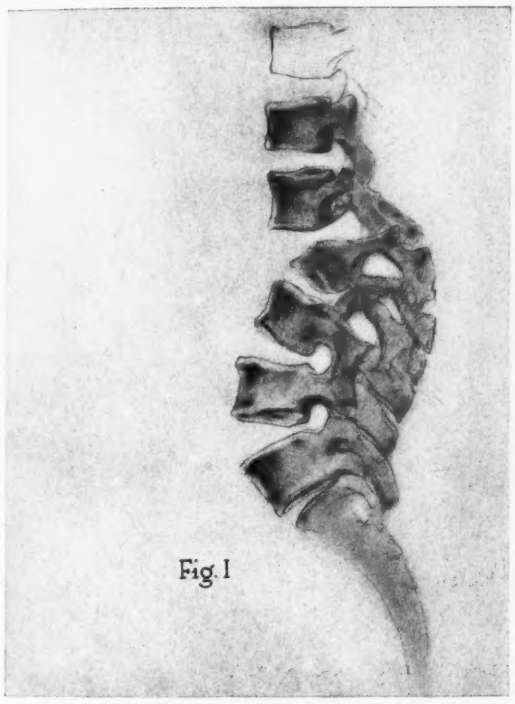


FIG. 1. Tuberculosis of the vertebrae.

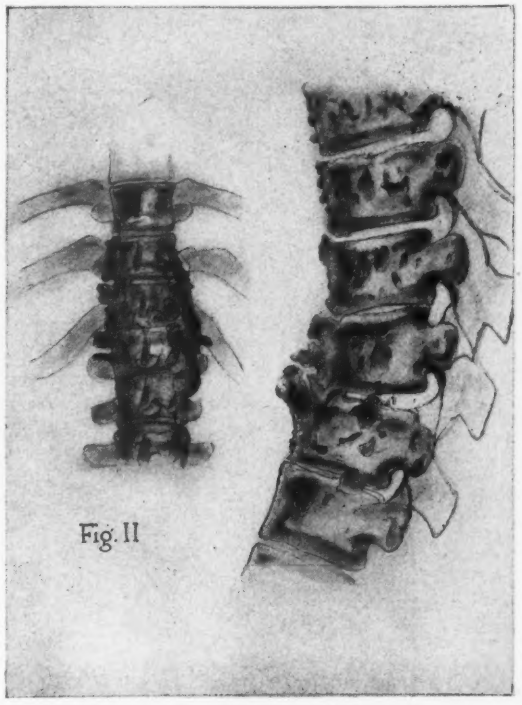


FIG. 2. Osteomyelitis.

Almost half a century has passed since Hadra presented his spinal operative procedure. There has been marked improvement in surgical technique, *x*-ray advancement and diagnostic accuracy which has allowed the subject of spinal and pelvic fusion to occupy a position which it properly deserves. In the interim there has been a tendency to become too enthusiastic about the possibilities of fusion which often created an unfavorable impression through misapplication, and lessened the value of true, sound, scientific accomplishment. It is the hope to preserve the usefulness of the fusion operation by proper selection of cases. This can only be accomplished by applying such procedure to correctly diagnosed and physically qualified patients. Spinal surgery has great possibilities but, no doubt, repeated failures due to enthusiasm or poor diagnostic

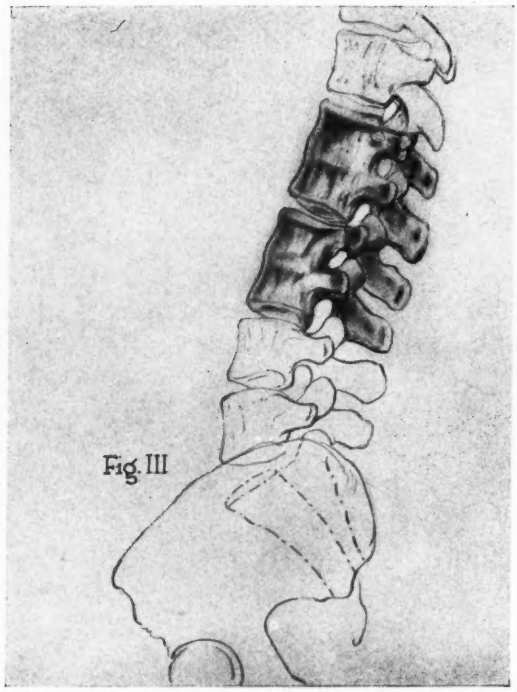


FIG. 3. Infection of vertebrae and intervertebral discs.

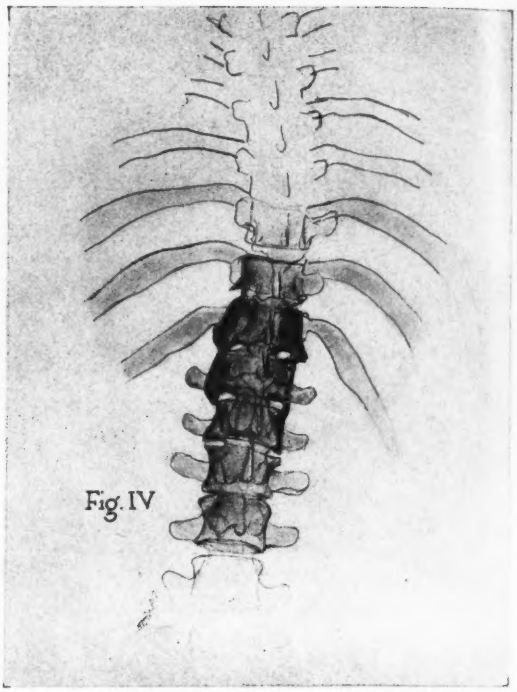
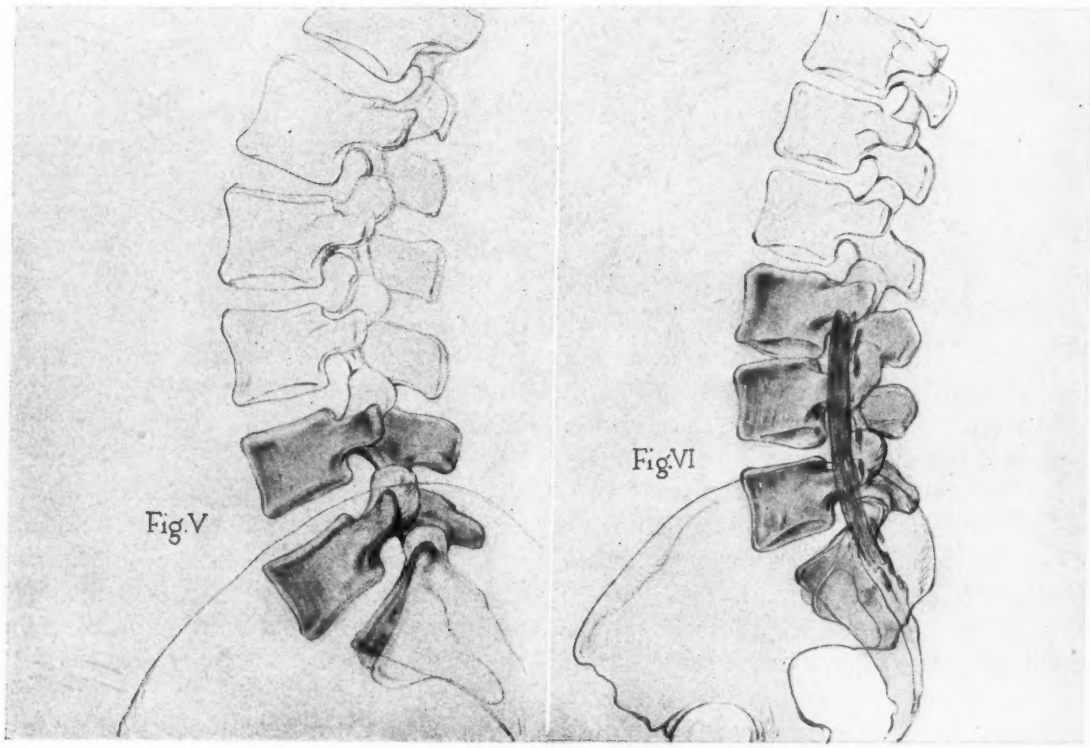


FIG. 4. Chronic arthritis.



FIGS. 5 AND 6. Spondylolisthesis.

judgment have discouraged many as to its usefulness.

A true understanding of any physiologic function is derived from observation of the normal as we are accustomed to see it. The spine and pelvis afford architectural stability combined with motion. The motion of the vertebrae or pelvic joints requires elasticity of structure, joint surfaces and coordinated neuromuscular control.

When any of these structures are altered by pathology or trauma, pain usually follows. Add to this referred visceral pain and the other many possibilities and it soon becomes evident that an accurate correct diagnosis is not always simple. Any why pain? It is for the relief of pain that practically all the patients seek surgical advice. Clinical observation shows that the majority of backache sufferers recover; few, if any, in this group succumb to the affliction. So it is pain relief they seek.

Examination of these cases over a period of years shows the physiologic reactions which occur. The natural forces present us with a formula to follow. If this is carefully observed, it can improve our surgical approach. Disease of the bony structures tends to fusion by ankylosis and even ligamentous invasion in an endeavor to stop motion and allow the individual to recover. There are degrees of this effort, to be sure, but the principle is constant. Trauma, in contrast, as a rule does not tend to bony fusion. Calcium may be deposited in the ligaments, but the true bony structures do not ankylose.

In congenital anomalies the body makes an attempt at correction by compensatory exaggeration, usually mechanical at a reasonably early age.

Intelligent observation and the application of these natural principles lay a good foundation for spinal and pelvic fusions. Since disease usually ankyloses the joints, advantage can be taken in such cases to expedite immobilization by operative fusion. Certainly there is no other operative reason for fusions. Many surgeons of large clinical experience regard such operative efforts to hasten healing of a diseased structure as unnecessary even here. If

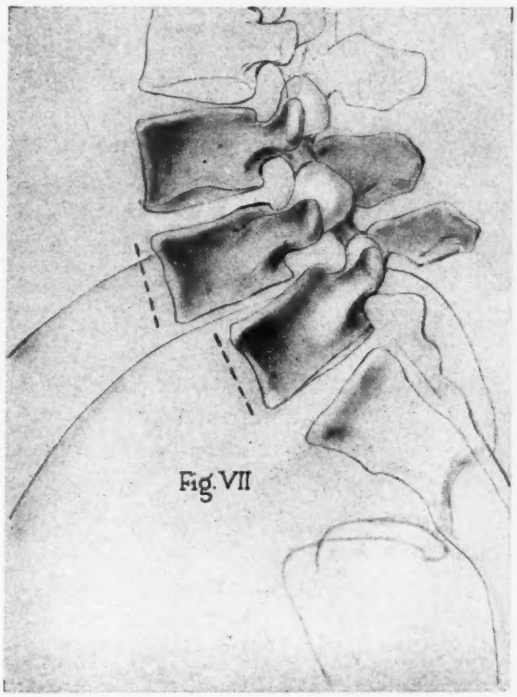


Fig. 7. Dislocation of fourth lumbar vertebra.

opinion is so divided on a process in which nature definitely points to fusion, how divergent must be the professional mind on traumatic lesions for which the natural process is restoration of normal function and not fusion.

Logical conclusions require correct premises. The premise in medicine is the diagnosis. This must be correct; then, if a case is placed in the fusion group, it will respond properly. What is conservative in one patient may be entirely wrong; the same may be true of radical measures. There are definite conditions of the spine or pelvis which demand surgical intervention—certainly that is not radical. There are others where non-surgical measures are used and accomplish nothing. One could hardly call that conservative. Therefore it is necessary to examine the diagnostic possibilities and try to formulate a procedure to relieve the patient of his disability.

The fundamental purposes of spinal fusion in all cases are usually: first, the

relief of pain and, secondarily, the maintenance of architectural stability with loss of motion. This means compensatory motion in most cases, and for this patients must be young adults. Old spinal joints are quite inelastic, becoming more so with age.

Perhaps it will be well to indicate possible etiologic factors of pain and convince ourselves of the small group among them to which fusion is applicable. The list is not all-inclusive but will, I believe, present the idea.

Congenital Deformities
1. Spina bifida of various degrees.
2. Vertebral malformations, body, spines, trans-process, articular facets, etc.
3. Spondylolisthesis.
4. Accessory ribs.
5. Muscular anomalies and defects.
6. Lumbosacral variations.

Mechanical Instability
1. Variation of leg length.
2. Scoliosis and associated lesions.
3. Foot disorders.
4. Posture.
5. Nerve root pressure by bony structures.

Infections
1. Syphilis.
2. Tuberculosis.
3. Arthritis:
   a. Acute.
   b. Chronic (all types).
4. Adjacent abscesses causing pressure.
5. Neuritis, herpes zoster, sciatica, etc.
6. Infantile paralysis.
7. Miscellaneous infections, as typhoid spine, actinomycosis, etc.
8. Osteomyelitis.
9. Myositis.

Tumors
1. Benign:
   a. Osteomas and other new growths.
   b. Nucleus pulposus protrusions.
2. Malignant:
   a. True bone tumors.
   b. Soft tissue tumors.
   c. Metastatic tumors.

Tumors of Adjacent Structures
1. Spinal cord tumors.
2. Spinal nerve tumors.
3. Pressure from extension, as pelvic malignancy hemangiomata, etc.

Metabolic Variations
1. Gout.
2. Hypothyroidism.
3. Loss of elasticity due to age.
4. Osteomalacia.
5. Vitamin deficiency, as rickets, etc.

Traumatic
1. Vertebral body fractures.
2. Spine and transverse process fractures.
3. Facet fractures.
4. Pelvic fractures.
5. Subluxation of sacroiliac joint, not completely reduced.
6. Sacral fractures.
7. Dislocation of vertebrae.
8. Sprains of ligaments and muscles.
9. Strains of ligaments and muscles.
10. Hematomata of spinal muscles.
11. Contusions of soft tissue.
12. Kummel's kyphosis.
13. Traumatic arthritis.

Miscellaneous
1. Malingering.
2. Neuroses.

Referred Pain
1. Pelvic and bladder pathology.
2. Abdominal pathology.
3. Renal pathology.

If one merely reviews the above diagnostic possibilities, it becomes quite clear how cautious one must be in recommending operative fusion for back pain. The frank, clear cases are rather easily identified but the obscure symptoms often offer a pitfall for operative advice. Who has not seen a case the diagnosis of which has clarified under prolonged observation? Truly fusion is not reserved for the so-called resistant cases. Many of these, in fact most of them, would not respond to fusion. Those cases

which respond to rest, recur on activity and again respond to rest, probably present the best prognosis, for this chain of events indicates a mechanical irritating factor

Figure 2 shows the exuberent new bone formation in an osteomyelitis of the vertebrae of the lumbar area. Again physiologic fusion.

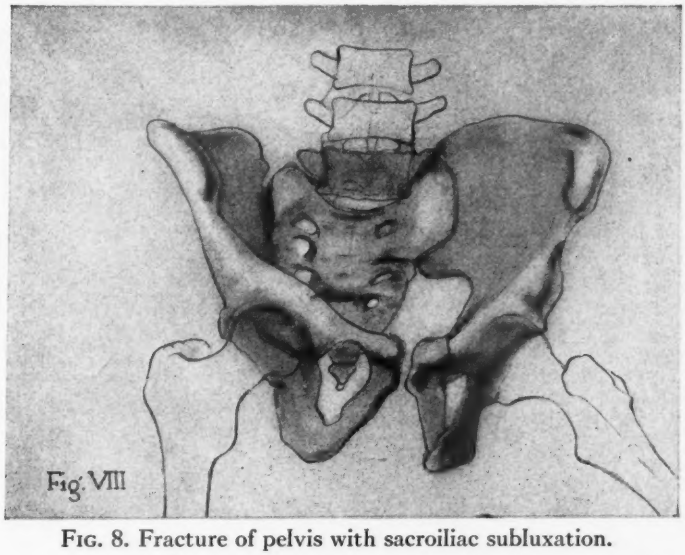


Fig. 8. Fracture of pelvis with sacroiliac subluxation.

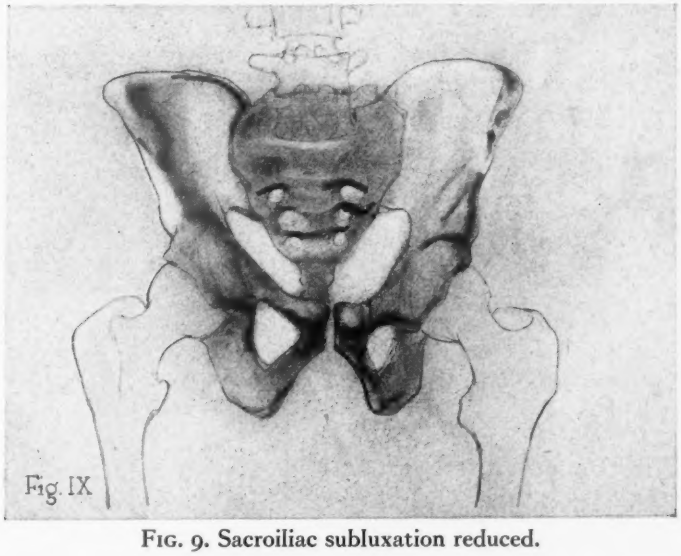


Fig. 9. Sacroiliac subluxation reduced.

relieved by rest, and fusion is a means of resting or immobilizing the affected part.

The illustrations presented here, drawn from originals, review the outstanding features of a few *x*-ray studies. The tuberculous spine (Fig. 1) represents a familiar arresting of disease by natural fusion. It is not necessary here to discuss the value of operative assistance in these cases. Hunchbacks have walked in history centuries before the spinal fusion.

Figure 3 is a vertebral infection involving the intervertebral discs as well as the adjacent vertebrae. A beautiful fusion has resulted here between the bodies of the vertebrae. This patient was recumbent for three years and is now ambulatory—an excellent non-operative result.

The fourth case is a type which may be seen daily in any clinic—the degenerative type of arthritis with the fatty medullary changes, the marginal prolifera-

tion and the ligamentous invasion. Here there is a natural fusion, slow, usually past middle age, but who has not seen one which was fused at some time, only to have the condition express itself in some other part of the spine or in many joints of the body. These changes occur in older people and in advanced cases no one would even consider fusion. In the earlier stages one must be watchful.

Figure 5 is of a case of spondylolisthesis discovered in routine *x*-ray examination. The patient had had no symptoms referable to the condition, although she was 32. Nothing was done. Figure 6 shows another case of spondylolisthesis which was observed for fourteen months. The pain and discomfort were progressive. This individual had a fusion done for relief of the condition.

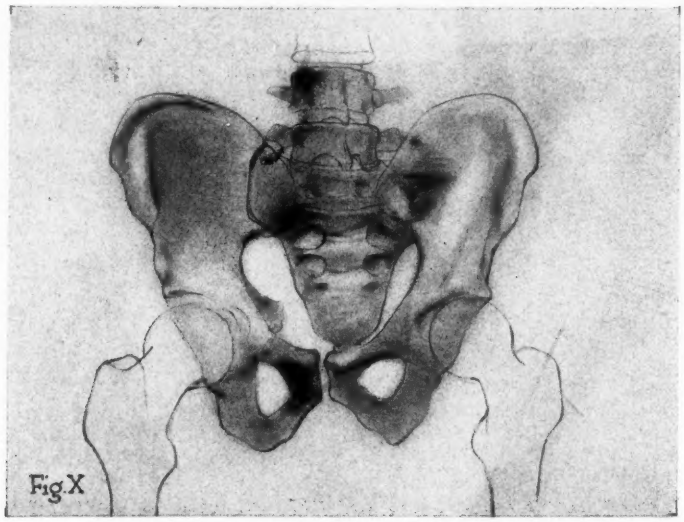


FIG. 10. Sacroiliac tuberculosis. Fused.

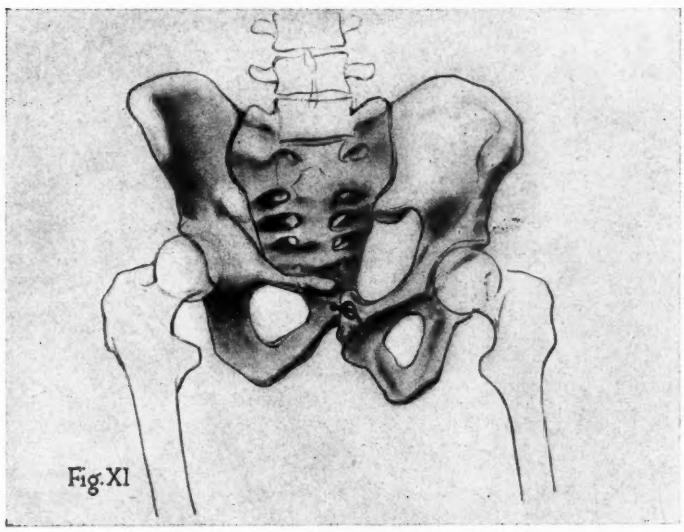


FIG. 11. Separation of ilium and symphysis. Fused.

Figure 7 illustrates anterior dislocation of the fourth lumbar vertebra. No spinal cord symptoms were present, but constant pain enabled the patient to walk only with difficulty. Spinal fusion with reduction of the dislocation brought relief.

The next case, shown in Figure 8, is interesting. Many fractured pelves show sacroiliac joint damage. Many present partial dislocations of varying degrees.

No doubt the majority spontaneously reduce when the traumatic force ceases. Careful investigation of the *x*-ray plates will allow early reduction of the dislocation if necessary. This is easily done (Fig. 9) by locking the femorae in internal or external rotation as the case may be and applying controlled force to reduce the condition. If this procedure were used more often, no doubt there would be fewer traumatic sacroiliac joints. This particular patient was relieved of severe pain by reduction. Six months later he was an oil field worker, completely free of any symptoms.

Figure 10 is merely one of sacroiliac fusion for tuberculosis, a justified procedure, we believe.

The next pelvic condition, Figure 11, was a severe injury to the pelvis with separation of the symphysis and the ilium. Since there was no improvement, a pelvic fusion was done. The patient now works as a laborer.

We have reviewed eighty-one case records of consecutive fractures of the spine. Since statistics can always be made to answer certain favored interpretations, it will be well to quote only a few facts. There were: one spinal fusion; four laminectomies; seventeen deaths, eight with paresis or paralysis; two hysterical hemianesthesia; and five patients with persistent pain symptoms. There was then about six out of eighty-one who were possible candidates for spinal fusion. This is a record to show the general result of conservative treatment of traumatic vertebral injuries of the severe type.

The milder types of injuries, muscular, ligamentous, hematomas, and the like, contribute even a much larger number of cases whose recovery is merely a question of rest and time.

In regard to the bony pelvic injuries, one hundred consecutive cases were examined. There was one fusion; six died from concurrent trauma; four had definite disability which would probably have been relieved by operative fusion. Although absolute accuracy is impossible, the number actually needing operative fusion is relatively small.

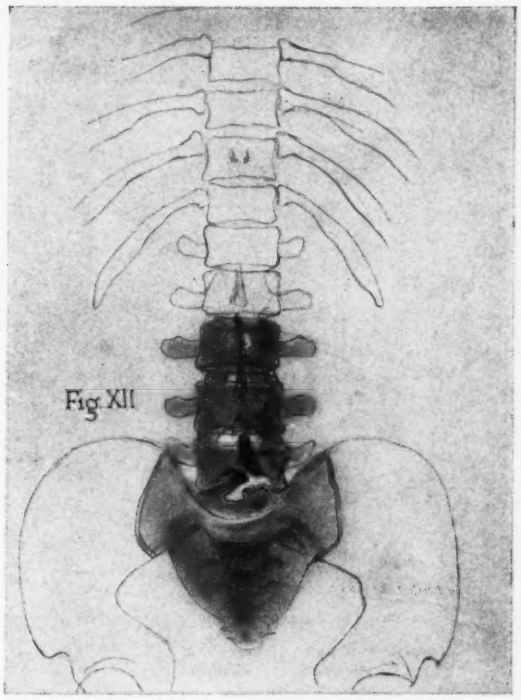


Fig. 12. Congenital anomaly of fifth lumbar and first sacral vertebrae.

The necessity of a correct diagnosis in the traumatic back is demonstrated in Figure 12. This man had backache for fourteen years. He had been at work and many times was treated for the condition. A sacroiliac belt was worn for four years and sacroiliac fusion was recommended. The pain was worse when he stood on his left foot and could be relieved by resting in bed. This happened to be one of the few cases of congenital anomaly causing a real disability. The distorted arch impinged upon the spine above. When the arch was removed, recovery was immediate. There are many such problems of diagnosis and many, more difficult. Had a sacroiliac fusion been done in this instance, the result would have been unsuccessful and a sound operative procedure would have received a demerit, the patient no relief.

The prominence of back injuries in industry and accidents requires rather special mention because many so-called injuries are often manifestations of pathol-

ogy and not trauma. Also occasionally among them are found unscrupulous persons who are malingerers. Frequently we can prove to our own satisfaction that the injury is assumed, but it is difficult to make the opinion a fact. All of us have seen many backaches disappear when legal settlements were made. It is a duty of the medical profession, however, to protect honest workers and see that they are properly cared for within the laws and customs of the community. Because of this situation it is apparently generally accepted that an amicable settlement should be made and the patient allowed the choice of a fusion operation if necessary. Patients so doing, as a rule, are more satisfactory for the surgeon to deal with.

SUMMARY

There is no doubt that fusion of the spine and of the pelvis is an accepted operative procedure which will give excellent results. Persistent sacroiliac pain relieved by rest can be given permanent surgical relief. The mechanical deficiencies of the spine, certain types of pathology of the spine, and selected traumatic conditions can be greatly assisted and comfort may be given to the patient. Before a patient is eligible for such operation, however, he should receive careful investigation and an accurate diagnosis. When he finally arrives on the threshold of spinal fusion, his age should be a determining factor; for the young and the old fusion operations are not preferable. The group of available patients, therefore, grows smaller and smaller until the number qualifying for this procedure is very small—insignificant as compared to the persons presenting themselves with backache. If one must err, let it be on the side of allowing too much bed rest for the patient and doing fewer fusions, especially in traumatic cases.

RUPTURE OF THE SUPRASPINATUS

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I HAVE long had the following convictions regarding shoulder injuries:

1. The shoulder has been, in the past, neglected by the medical profession including anatomists as well as surgeons.

2. The shoulder joint is apparently simple, but is really very complicated, and this is perhaps one reason why the clinical conditions which affect it have remained obscure, for both its anatomy and its functions have never been clearly understood, and hence have not been taught to medical students.

3. Other reasons for the neglect of this joint are that shoulder lesions are seldom of a serious character or of a permanent nature. They cause no limp or deformity as do lesions of the joints of the lower limb; the chief symptom of which patients complain is pain and there are few obvious signs commensurate with the complaint of suffering; swelling of the joint structures is difficult to detect. Surgeons have until recently found it more interesting, and perhaps more important to focus their attention, study and discussions on other parts of the anatomy as the head, thorax, and abdomen, injuries to which cause serious conditions and even death. Disabilities from joints of the lower limb, which unlike the arm, is subject to many fatal, serious, or obviously deforming injuries, attract the attention of the laymen as well as physicians. Anyone recognizes the limp of a patient with a stiff hip joint, but we do not recognize the stiff shoulder joint as it passes us on the street, and many a physician has failed to recognize one in a cursory office examination.

4. The advent of the workmen's Compensation Act has brought a great change, for now total disability on account of a shoulder lesion costs as much as does such disability from any other lesion. Formerly the patient simply quit work at his own expense and could be treated without any detailed diagnosis by any method of treatment which the physician was equipped to give. Since most shoulder lesions recover in time by nature's own methods, it made very little difference what method was chosen by the doctor. Thus each form of treatment had its advocates who knew that most patients had recovered while under treatment by their various splints, analgesic drugs, massage, passive or active exercises, baking, liniments, acupuncture, cautery, hydrotherapy, the various forms of electric treatment, and the more modern applications of light and heat. Yet all of these ardent advocates had to admit the occurrence of occasional obstinate cases.

5. Treatment by surgery has been relatively neglected, because successful surgery requires not only exact diagnosis, but a detailed knowledge of the anatomy and normal motions of the shoulder and an understanding of the pathology. Moreover, ill-advised surgery may do harm. Nevertheless, surgery will be done more and more now that each shoulder patient who lingers in recovery has become a great expense to industrial insurers, and is no longer merely a patient who will not work because he complains of pain in the shoulder. Even the laymen employed by these companies begin to demand an exact diagnosis and more efficient treatment. The manager, the treasurer, the statistician and the claim agents ask questions.

Times have certainly changed, and though it is probably true that in many industrial clinics, shoulder lesions are still neglected and treated by physiotherapy without careful study or diagnosis, I feel

sure this is not true in the better clinics. I receive letters from doctors in various cities telling me that they have made early diagnoses, and have operated upon cases of complete rupture of the supraspinatus tendon—the one lesion of all others in which I believe prompt diagnosis and operation are most important. Unless these patients are operated upon, they never completely recover good use of their arms.

Only a minority of other obscure lesions of the shoulder absolutely require surgery according to our present lights, but the field for surgery is broadening, as we become more exact in our diagnoses. It has been my aim for years to make the medical profession supraspinatus-conscious, so that these cases may be promptly recognized and treated surgically. I hope that the publication of this article will help toward that end.

With the above convictions in mind, I have written what follows as if I were addressing a young surgeon who had recently been appointed to take charge of the clinic of an insurance company or of the injured employees of a large industrial plant, assuming that he had come to me for what points I could give him to enable him to get good results in his cases of injured shoulders. I shall address him frankly, referring to my book "The Shoulder" when necessary, and hoping to stimulate him to study it thoroughly and then to try to learn more from his own experience, whether this proves confirmatory or contradictory to what I have written.*

ADDRESSED TO AN INDUSTRIAL SURGEON

You will undoubtedly see many patients who complain of sore shoulders, and you may well be astonished by the number of dollars which are paid by your company as compensations for these injuries. It is up to you industrial surgeons to help diminish this figure, even though your own salaries will not be increased if you make some discovery that will cut this mighty sum in half! I offer suggestions of possible ways in which you can help in this problem.

First, ask your statistical department for a list of all cases of shoulder injury occurring in the year 1936, in which the patient did not return to work within a year. Obtain their history folders and separate these into two piles, the first containing the diagnoses of wounds, burns, infections and fatal injuries, and the second containing the histories of all other injuries of the shoulder.

I have no suggestions about the first pile; you may return them to your statistician and ask him to help you study the remainder, for you will, I think, find that most of these were instances of rupture of the supraspinatus, even though complicated by fractures, dislocations, circumflex paralyses, etc. It is in this group of cases that you will find evidence to indicate that prompt and efficient treatment might have saved your company many thousands of dollars. Each case will have already cost perhaps $3,000.00, and, if your company is a large one, you may have to multiply this by a good sized figure. If you had twenty cases in 1936 the total cost would be about $60,000.00. At any rate the loss from these cases was probably more than double your salary.

Now, you will ask why I think that the second pile will be largely composed of cases of rupture of the supraspinatus. In the first place, the lapse of a year without recovery since the accident eliminates most of the other known shoulder lesions. Cases of simple strains, contusions, minor injuries, acute traumatic bursitis, most cases of calcified deposits, and even most instances of the more obstinate periarthritis, which I choose to call "tendinitis," recover in less than a year. Even malingerers and neurotics have usually been eliminated by that time; in fact, if a patient had been labeled in either of these classes and came to me after a year, my first presumption would be

* In the following text, the page numbers cited as references relate to Codman, E. A. "The Shoulder." Figures 1 to 8 in this article are reproduced from the same source.

that he would present an instance of unrecognized complete rupture of the supraspinatus. There are still many doctors who have never heard of this lesion, and many more who have never recognized a case of it; these doctors are apt to use the labels of "neurosis" and "malingering." Study the cases so labelled carefully; if they have atrophy of spinati, they are more than likely to be the cases which we are seeking. One might say that a common symptom is "to be accused of malingering."

Another question will be to ask my reason for telling you to put the histories of cases of fracture and dislocation in your second pile. Because I believe that cases of uncomplicated dislocations or fractures of the shoulder recover within a year. If, in any given case the patient is not at work within a year, the most likely complication to cause this delay is rupture of the supraspinatus, or, what I consider the same thing, evulsion of its facet of insertion. If the facet alone is torn away by the tendon, the joint communicates with the bursa, but in cases where a fracture of the tuberosity in the usual triangular form takes place, the bursa remains intact with its base still on the tuberosity. Two raw opposed bony surfaces remain in partial contact in this usual fracture, and union will take place. A retracted facet, however, has no chance of bony union because it can make no contact, so that the conditions are essentially the same as when the tendon is ruptured or evulsed. Bursa and joint will be in communication and healing will not take place.

I find that long delayed recovery from either fractures or dislocations of the humeral head is usually due to this complication, and I plead for prompt operation when the *x*-ray immediately after the accident or after reduction of the dislocation or fracture shows a small retracted facet. (Page 88.) I believe that after the reduction of a dislocation the patient should be made to raise his arm in internal rotation in order to test his ability to do so. (See pages 290 and 393.)

I have also asked you to put cases of muscle paralysis in the second pile because I have several times seen circumflex paralysis accompanied by complete rupture of the supraspinatus. The latter had not been recognized, for the symptoms had been attributed entirely to the paralysis. This mistake is more readily made because in these cases, owing to the circumflex paralysis, sensation in the joint is partially destroyed and the pain caused by the elevation of the arm is lacking. The deltoid does not function and therefore the patient cannot attempt to elevate the arm and cause the bare tuberosity to gain a fulcrum on the acromion. These cases present the picture of "dropping shoulder." I consider them so important that I have given a whole chapter (see page 42) in my book to them. I have found that in pure circumflex paralysis, the patient can feebly elevate his arm by the use of the supraspinatus alone, if the latter is uninjured. If he cannot do so, exploration of the bursa should immediately be done to determine whether or not the supraspinatus has been ruptured. Difficulty arises in this diagnosis because we occasionally see cases in which circumflex paralysis is only a part of a root palsy of the Erb type. In such cases, exploration is seldom necessary, for we can make the diagnosis if we also find paralysis of other muscles than the deltoid which have nerves arising from the brachial plexus, especially those supplying the rhomboids, the clavicular portions of the pectoralis major, and the biceps.

A clear description of the character of the injury is helpful, too. Blows or falls of a violent nature, which depress the whole shoulder in relation to the body, are the ones which tear the upper roots of the plexus where they emerge from the cervical spine, while pure circumflex paralysis is usually due to falls on the elevated arm accompanied by dislocations of the head of the humerus. (Page 354.) In the latter case the circumflex alone is stretched by the dislocated head of the bone, and temporarily paralyzed, although occasionally

immediate spontaneous reduction of the dislocation occurs. Such paralyses are seldom permanent although they may continue for many months if the patient is allowed to be about with the arm hanging down unsupported. This constantly stretches the paralyzed deltoid, and also puts an unnatural strain on the uninjured supraspinatus, tiring it out. (Pages 385 and 482.) If the arm be supported in elevation these patients promptly recover. Root paralyses, on the other hand, are usually permanent, although, fortunately, they are rare.

There will also be some cases in which rupture of the long head of the biceps has been detected, and this lesion blamed for the more severe symptoms which were in reality due to a simultaneous rupture of the supraspinatus. The two injuries frequently occur from the same accident. In my opinion, isolated rupture of the biceps is a relatively trivial injury. In most cases the rupture occurs in the tendon at the upper edge of the glenoid labrum, and in such cases the muscle belly at once pulls the tendon down into the groove out of harm's way. Nothing is left to interfere with the motion of the joint, except that one element is lacking which tends to hold the joint in place, and to keep the head of the bone from riding forward. (Pages 242–243.) This function of the biceps is especially needed when the supraspinatus is also completely torn, in which case two important restraining factors are destroyed and there is a definite palpable forward displacement of the head of the bone. (Page 290.) The symptoms in such cases are somewhat more pronounced than when the supraspinatus alone is torn, and much more pronounced than if the biceps only has given way.

Gilcreest has called attention to the fact that partially torn biceps tendons may give more painful symptoms than do those which are completely ruptured. The frayed tendon irritates its sheath and does not run smoothly during motions of the shoulder. This pain causes spasmodic restriction and, perhaps later, adhesions of the tendon to the sheath. He records a case in which only a fine strand of tendon remained in the groove and caused disability which was completely relieved by excision of the strand and suture of the remaining portion of the tendon to the coracoid.

You will probably find a few cases in which the diagnosis was "acromioclavicular arthritis" or "dislocation of the acromioclavicular joint." Beware of attributing very disabling symptoms to these diagnoses unless you can also demonstrate rupture of the coracoclavicular ligaments by finding a palpable gap between the clavicle and the coracoid. In such cases operation is needed, while pure acromioclavicular subluxations can usually be treated conservatively. Never forget that many laboring men have enlarged acromioclavicular joints without ever having had a symptom from them. Such enlarged joints may easily be bruised and become sore and tender, but they do not interfere much with the motions of the true shoulder joint, nor do such symptoms often persist for over a year. If these patients complain that they cannot raise their arms, remember that they may have also injured their supraspinati and don't fail to look for the characteristic symptoms.

The type of injury which causes acromioclavicular dislocation is quite different from the type which causes scapulohumeral dislocation. A sudden blow, usually caused by a falling object, on the tip of the acromion and not borne in part by the outer end of the clavicle, is the usual one in the former, while forced elevation of the humerus, either active or passive, is the usual cause of anterior scapulohumeral dislocations and of ruptures of the supraspinatus.

However, it is very difficult to get clear histories in shoulder injuries, so that one must accept them guardedly. "Fell and struck my shoulder" is the usual one. All the patient really knows is that he fell and his shoulder was hurt. He cannot

attribute it accurately to any of these other more likely possibilities:

1. He suddenly threw up his arm to save his balance.

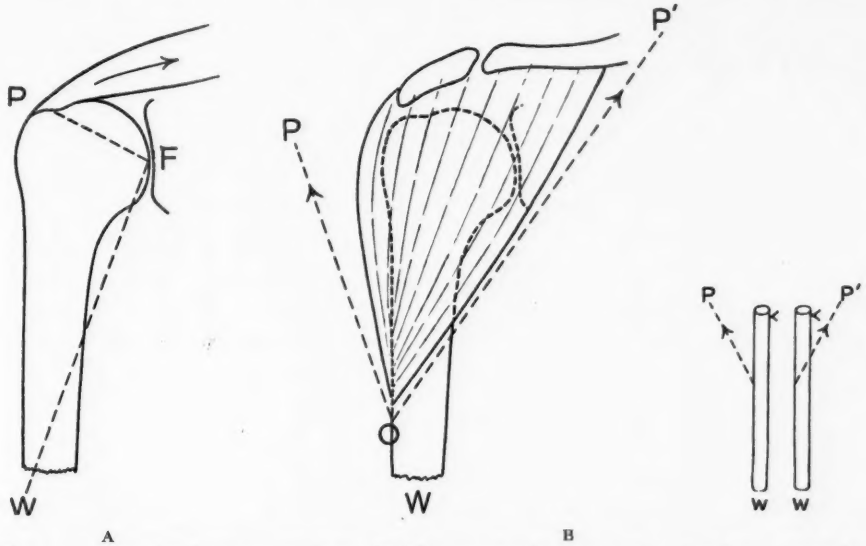

Fig. 1. A, showing how power is applied by supraspinatus. B, showing how power is applied by deltoid.

2. He grasped something as he fell and made a violent effort with that arm.

3. As he fell, he twisted the arm under him.

4. Force was transmitted to his shoulder by his outstretched arm and hand in trying to break his fall.

I defy any man to fall and strike his shoulder unless he is holding something tightly under his arm. The arm is instinctively thrown up to regain balance if the fall is backward or to the side, and if it is forward it is at once raised to fend off the ground. The inertia of the arm is slight compared to that of the body and it can be elevated quicker than the body can fall. Moreover, as explained on page 9 of "The Shoulder," it would be very difficult to strike a blow on the articular head of the humerus. In this limited space, I cannot repeat the long argument I have made in chapters 9 and 10 for my belief that hyperelevation of the arm, combined with internal rotation, is the cause of most fractures of the tuberosity and of the humeral head. Anterior dislocation is, I consider, caused by falls in which the hyperelevated arm is rotated outward and carried into the pivotal position, so that it is dislocated while momentarily erect, and then as the patient rises it falls into the position in which we usually find it, internally rotated at the side. Backward dislocations are usually caused by twisting the arm in internal rotation behind the back. Ruptures of the supraspinatus frequently accompany anterior dislocations, or may occur from sudden elevation of the arm to regain balance when a patient falls backward or to one side. To raise the weight of the arm suddenly against gravity puts a tremendous strain on this little tendon, before the angle of the arm in relation to the body has increased enough to permit the deltoid to help with its power. (Fig. 1.) Study the figures in the chapters on fractures and dislocations, and compare the descriptions of the accidents on pages 140 to 144, but beware of the patient's own statement that "he fell and struck his shoulder," for this rarely happens. An eye witness's evidence is more likely to be correct.

Your pile of histories will therefore probably contain some instances of the following mistakes in diagnoses.

1. Unrecognized isolated ruptures of the supraspinatus.

2. Those associated with fracture of the head of the humerus.

3. Those accompanying dislocation of the head of the humerus.

4. Those which have passed as circumflex paralysis.

5. Those which have passed as neurosis or malingering.

6. Those that accompanied ruptures of the long head of the biceps.

7. Those that were misdiagnosed as acromioclavicular arthritis.

8. A few cases will appear under miscellaneous misdiagnoses if your examiners have been too busy or somewhat careless. For instance, "arthritis," "bursitis," "neuritis," "rheumatism," are no longer diagnoses. They are just words which doctors once used to satisfy their patients, not the names of definite lesions requiring definite treatment. I will admit that we still must use "arthritis" to indicate inflammation of a joint or joints, but to be reasonably valuable it must be preceded by one or more descriptive adjectives, such as gonorrheal, tuberculous, syphilitic, traumatic, generalized hypertrophic or atrophic, etc., but these terms seldom apply to a shoulder joint. I have rarely seen a case of adjectiveless "arthritis" confined to one shoulder. Generalized hypertrophic or atrophic arthritis may affect one or both shoulders, but as this joint is not essentially weight bearing and the joint cavity is large, it is rarely deformed by inflammation. Usually much milder symptoms occur in the shoulder than in such joints as the knee, which are difficult to rest. If you have a case labelled "arthritis," and no other joints in the patient's body show more evidence of "arthritis" than does the shoulder, I am ready to say that this is probably an instance of complete rupture of the supraspinatus. I will say the same thing if you have a patient with so-called "bursitis" who is not well after a year has passed (even if the word before it is subdeltoid or subacromial). A "bursitis" which prevents a patient from working for a year after a fall was probably caused by a rupture of the supraspinatus. Bursitis does not persist without a cause, and much the same thing can be said of the cases labelled "neuritis" and "rheumatism" of the shoulder. "Bursitis," like "peritonitis," still occurs, but nowadays we are not satisfied with the words without knowing the exact cause in each case.

If you have now withdrawn from your pile of histories these eight classes of suspicious cases, how are you going to tell whether these patients really had complete ruptures of their supraspinatus tendons. I must ask you now to study carefully Chapter v in "The shoulder" in which the symptoms and signs of this lesion are described. I can only give you here a list of the eighteen criteria which should be present to establish the diagnosis. (Table 1.)

TABLE 1

CERTAIN CONDITIONS, SYMPTOMS AND SIGNS WHICH INDICATE COMPLETE RUPTURE OF THE SUPRASPINATUS TENDON AND WHICH SHOULD BE PRESENT WITHIN TWENTY-FOUR HOURS AFTER THE ACCIDENT

1. Occupation—labor.
2. Age—over 40.
3. No symptoms in shoulder prior to accident.
4. Adequate injury—usually a fall.
5. Immediate sharp, brief pain.
6. Severe pain on following night.
7. Loss of power in elevation of the arm.
8. Negative *x*-ray.
9. Little, if any, restrictions when stooping.
10. Faulty scapulohumeral rhythm.
11. A tender point,
12. a sulcus, and
13. an eminence
14. at the insertion of the supraspinatus,
15. which cause a jog,
16. a wince, and
17. soft crepitus, as the tuberosity
18. disappears under the acromion when the arm is elevated, and usually also as it reappears during descent of the arm.

When you have studied Chapter v, send for the patients whose histories you have and examine them for these criteria. I am sure that you will find that these signs are present still in the majority of the patients. although probably not in their histories.

When you have carefully followed my directions thus far, if you do not find I am correct in this statement, do not bother to finish this article. If I am wrong in this, I am more than likely to be wrong in any

other statement that I make in the remainder of this paper, which will be mainly devoted to trying to help you to spot these

Fig. 2. Position of hands for examination of shoulder. The left thumb lies along the depression below the spine of the scapula and the tip of the forefinger is just anterior to the acromion. The other three fingers cross and hold the clavicle. Thus the shoulder girdle is firmly held and any motion of the scapulohumeral joint is at once detected.

cases immediately after their accidents, and to tell you how to prepare yourself to cope with them surgically. When you have examined a few of these old cases in conjunction with the study of the fifth chapter of my book, you will find that the diagnosis in these typical cases is easy. The mere fact that the same symptoms have persisted for more than a year, unchanged by treatment, goes a long way in making the diagnosis. Once you have mastered the method of examination shown in Figures 2 and 3, you will find it a great help, and will learn to locate the characteristic tender sulcus and to feel the soft crepitus. Your great difficulty will come in the fresh cases, for some of the symptoms are similar to those in other conditions which get well of their own accord within the first year, particularly those of partial rupture of the supraspinatus, in which the base of the bursa is not torn through.

If you are in earnest in this matter, and have convinced yourself by such a study of the past cases in your clinic that supra-

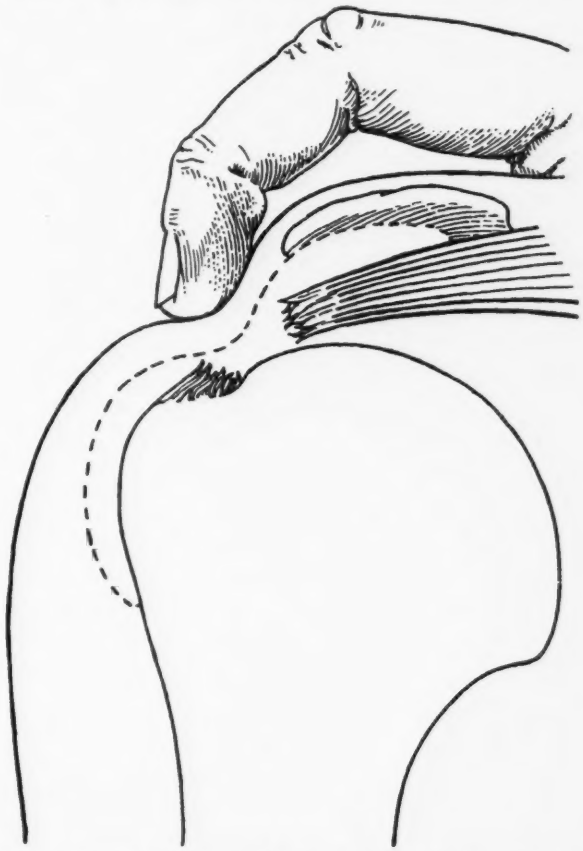

Fig. 3. Tip of finger pressing on eminence and on sulcus. The plane of this diagram is halfway between the coronal and sagittal. It is, perhaps, the most important diagram in the book ("The Shoulder") for the reader entirely to understand, for it is the ability to put the finger in this position which enables one to make the clinical diagnosis of rupture of the supraspinatus tendon. The dotted line represents the contour of the bursa. The sulcus lies immediately under the tip of the finger and the eminence just external to it. Therefore, as explained on page 148 of "The Shoulder," the tender point in a case of rupture is represented by a depression, but in cases of calcified deposit, by an eminence at the corresponding position.

spinatus injuries should be detected soon after accident and promptly operated upon, I want to urge you to do some careful dissections of fresh shoulders at autopsy before attempting to operate on a patient. You may have had A in anatomy at the medical school, you may be an experienced and clever surgeon, but you will find the shoulder a puzzling joint and experience much difficulty in telling one tendon from

another, and much more in bringing the appropriate ends together, if the case is a long-standing one. Had I one of these ruptures I would prefer to be operated on by a recent interne who had made dissections of the shoulder, than by any of the distinguished surgeons of the country who had not refreshed their knowledge of the local anatomy. So it is advisable to make at least a few dissections after the manner described on page 24 of "The Shoulder" before attempting these operations.

It does not require much practice in dissection to learn how little can be seen through an exploratory incision in the normal bursa. (See pages 24 and 232.) When the little wound is held open, even if your assistant manipulates the arm correctly, the most that you can see is the uniformly rounded colorless surface of the base of the bursa. You can inspect practically the whole base from its inner to its outer edge by rotating the humerus, and, as the arm is carried forward and backward, you may see most of the vertical diameter of the base. If the bursa is normal, the whole base is a homogeneous, smooth, white surface, but this surface is opaque and covers approximately a 2 inch circle beneath which are the structures shown in Plate 1, close beneath the surface, but quite invisible, unless the base of the bursa is torn.

In operating, when you have made the exploratory incision and find the bursal base intact, let me urge you to back out at once. You may do more harm than good by exploring further in most cases. There is one exception, i.e. if you suspect that the biceps tendon has been torn. Then you may incise the upper end of the groove and inspect the tendon.

Although the base of the bursa is homogeneous-looking, you may learn something more with the tip of your finger, for one can palpate the edge of the tuberosity and also the bicipital groove. Keep in mind that the groove is very nearly in line with the flexed forearm. If the groove is empty, it means that the tendon has been retracted downward and if it seems best, you can hunt for the retracted end, bring it up, and attach it to the inner head of the biceps. If you find it split or frayed, follow Gilcreest's method. However, you must remember that in cutting the tissue over most of the groove you are dividing the tendon of the pectoralis major.

If you find that the base is reddened at the edge of the tuberosity, do not be intrigued into exploring farther. The redness probably indicates that some of the fibers are ruptured beneath the base of the bursa. However, if there is a calcified deposit shown in the *x*-ray, you may incise with the point of your knife in the center of the red area. (See page 233.)

The exploratory incision is safe and simple in the hands of any clean surgeon, if he will quit when he finds the base of the bursa intact. It is as effective as a larger cut could be because lesions of the bursa seldom occur in any part except in the portion so readily made visible through the exploratory incision by rotation of the arm. When an old complete rupture is found, however, its suture is a really difficult matter unless the operation is done soon after the accident, before retraction and partial cicatrization of the edges have taken place. I have seen a surgeon who was a good general operator, sweat for two hours over this operation simply because he was unfamiliar with the anatomy and pathology of the condition. I have done so myself, many times. Even with the great interest and considerable experience I have personally had in these cases, I find the long delayed repair of extensive tears extremely difficult and sometimes have been obliged to abandon entirely the attempt to complete them. The early cases would not be difficult for any surgeon to do; a harder task is to teach the practitioner to recognize them or at least suspect them, immediately after the accident.

In a case of rupture of the supraspinatus, you may see any of the structures shown in Plate 1 through the rent in the base of the bursa. You will certainly see the sur-

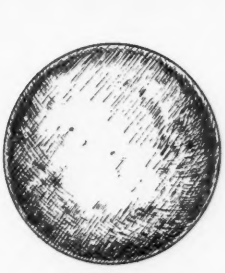

Fig. I

Fig. III

Fig. II

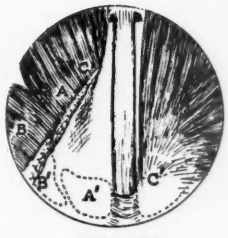

Fig. IV

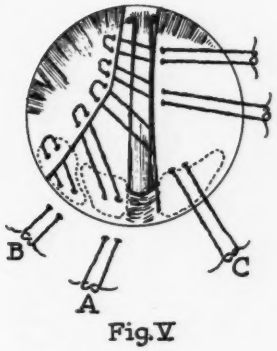

Fig. V

Plate I. These figures are so arranged that the small circles may be cut out and superimposed in turn on the larger figure. To avoid confusion no effort has been made to sketch in the operative incision in the skin or in the deltoid. The circles illustrate what may be seen by a combination of retracting the lips of the incision and rotating the shaft of the humerus on its long axis, although the whole field in each circle cannot all be seen at one particular moment. In these figures the humerus is externally rotated in relation to the scapula.

Fig. I. On opening the bursa in a normal shoulder, the base of the bursa is smooth, white, and glistening. Beneath this base, which is opaque, are the insertions of the short rotators, shown in Figure II.

Fig. II. The terminal portions of the muscles become tendinous and are inserted at their facets in the anatomic neck of the bone, on top of the greater and lesser tuberosities. These facets are indicated in the figure by dotted lines. The fibers of the infraspinatus are inserted more or less superficially to those of the supraspinatus, but the fibers of all the tendons blend to such a degree with one another that practically their junction cannot be seen, nor can they be clearly separated.

Fig. III. When all the muscles and tendons have been removed, the head of the humerus and the facets of insertion may be seen, with the long head of the biceps crossing the articular surface and descending into the bicipital canal. In some cases when all the tendons are torn and retracted this appearance is found as soon as one opens the roof of the bursa. The last patient in whom I did an exploratory operation presented this appearance, and the tendons had retracted to such a degree (in two and one-half months) that no attempt at repair could be made. The biceps tendon was strong and functioned well.

Fig. IV. The appearance which is found in most cases of complete rupture of the supraspinatus. Usually the subscapularis is not involved, but the supraspinatus and infraspinatus are often both torn. I have never found these two tendons separated from one another. They retract outwardly together, the infraspinatus pulling the supraspinatus, somewhat downward as well as outward, the point C being pulled outward but also obliquely upward. It is well therefore to begin closure at the outer angle, uniting B to B′, A to A′, and C to C′, as indicated in Figure V. One must realize that in old cases the falciform edge disguises the lines of division between the tendons and this edge should be pared off, down to sound tendon.

face of the articular cartilage; you will probably see the long head of the biceps, although you may have to retract the edge of the subscapularis to do so. The frontispiece in "The Shoulder" shows very well the appearances found in old cases, but in fresh ones the structures are much more easily recognized and therefore more easily repaired. In a recent case, the torn ends of individual fibers could be demonstrated and it would have been almost possible to suture them individually with a microscopic needle. It was very easy to approximate the torn ends. Usually these cases are not recognized early. They are only "office" patients and are given "treatment" and told to come again, but in this case the patient made his own diagnosis and came directly to me.

The history of this patient also illustrates other interesting points both scientific and social.

In May 1935, a sturdily built, healthy Irish laborer of 48, with no history of previous injuries, diseases or operations, fell off the step of a railroad car in which he had been employed as a cleaner, and injured his right shoulder. He had the typical symptoms of a complete rupture of the supraspinatus, but they were not recognized as such by the doctor employed by the railroad company, who would not admit serious disability and insisted that the man continue to work or lose his job. Palliative treatment did little good, and the same symptoms persisted month after month. For the sake of his wife and children he kept at his work, which was not difficult and seldom required the use of his right arm at levels above the shoulder. He found it possible to use the left one instead.

About a year after this he met a friend on whom I had operated successfully for the same injury several years before. This friend recognized the familiar symptoms and brought the patient to me. I made a positive diagnosis, and advised, but did not strongly urge, an operation, because so much time had elapsed and the patient had to support his family. The company by which he was employed would assume no responsibility. The patient continued to work and his symptoms persisted, especially the painful jog. I saw no more of him until December 23, 1937, when he came in again to report a similar condition in his other shoulder.

On the day before he had been holding up a car window with both hands (an effort which we all know justifies expletives) when the typical painful jog occurred in the injured shoulder (right). He winced and had the "down-it-will-go" reflex so strongly that he let go with his right hand and thus suddenly threw all the strain on his uninjured elevated (left) arm. He felt a sharp snap in that shoulder with the typical pain and immediate inability to raise the arm. Again the company doctor did not recognize what had occurred, accused him of lying and would not admit any disability.

Fortunately the patient this time made his own diagnosis and came to me the next day asking to be operated upon. I telephoned the doctor, told him my diagnosis and strongly urged operation. He told me he had found no evidence of injury, that the patient was a liar and he could assume no responsibility for the payment of bills if I chose to operate; he would need advice from the home office in Chicago. Knowing what delay this would mean, the patient and I decided not to wait, for if this second shoulder turned out to be as badly injured as the first, he would never be able to support his family.

Then another trouble arose and consumed more days before I could get a place in a hospital for him. A charitable hospital would not take him because he was an "industrial case"; a pay hospital would not take him because no one would guarantee payment of his board and he had no money of his own.

Like the making of the world, all this took seven days and I used all the authority and influence I possessed to say, "Let there be a bed for this man." Finally I had to personally guarantee payment for a bed in the Baker Memorial Hospital and get my recompense in the chance to see a really fresh rupture of the supraspinatus, for the patient will have little hope in suing the common carrier over whose accidents the Massachusetts Industrial Accident Board has no jurisdiction. His wages, which were only fifteen dollars, would hardly pay legal expenses even if collected.

The findings at operation showed a perfectly fresh tear in a good strong looking tendon with a portion evulsed from the tuber-

osity almost exactly as depicted in the frontispiece of "The Shoulder," except that there was little retraction and there were no falciform edges.

His convalescence was normal and the relief of pain so satisfactory that when good use of the left arm returned, he wanted to have the right shoulder operated upon, although I could not promise much after the lapse of two years since the original injury. At operation on May 23, 1938 I found a typical old rupture with falciform edges, the extent and appearance of which was almost identical with the one shown in the frontispiece. Here in one patient I was able to see the change from the early appearances in such cases to the late ones. It was most satisfactory for it had taken me years to interpret the observations made in the dissecting room so that I could logically reconstruct what the findings would have been immediately after the rupture of the tendon. I had been correct except that I had not realized how much of the retraction had been outward rather than upward. Thus the observations made at the first operation on this patient enabled me to do his second operation much more satisfactorily by the method to be described later in this article.

On September 1, 1938 the patient was able to work, but could not even get his old job back. He is suing the railroad in the hope of getting something to pay a part of his hospital bills.

This case was most instructive from the point of view of anatomic and surgical science, but it is of perhaps more value as a study of social science in relation to the cost of medical care. It is an instance of (1) the inefficiency of our medical organization; (2) our lack of a method to advertise new medical knowledge for the use of the practitioner; (3) the legal helplessness of employees of interstate common carriers; (4) the unsettled financial problem in hospitals; (5) our need of some agency to distribute patients to those qualified to treat them; (6) the importance of giving expert services to the immediate treatment of industrial accidents instead of in court years later; (7) the need of clinical research combined with statistical analysis of the records of industrial cases; and of many other social problems. Each of these headings would survive committees of Congress without solution, but a great step toward the ultimate solution of all of them would be the introduction of the "end result system of organization" into hospitals.

Let me point out to you how confusing it is to recognize these structures through the small exploratory incision if you are not thoroughly informed about their relative positions, crowded as they are into an area the size of a silver dollar. You can see only a portion of this area at a time, and this very portion is being moved one way or another as your assistant rotates the arm. You are looking through a very small wound and all the surroundings are covered with towels. I hope that you will study this small field until you don't have to think twice about such questions as the following ones:

If you are operating on the left shoulder and are standing cephalad to the arm and you want to see the portion of the bursa over the subscapularis, would you ask your assistant to rotate the arm in the same direction as you would the right arm if you were operating upon that and standing caudad to it? Would you expect him to rotate that arm so that the floor of the bursa would rotate to the right, from your point of view or from his? As right and left refer to your point of view, not to his, you will avoid confusion by asking him to rotate outward in both instances, for outward rotation of the arm brings the subscapularis facet under the incision in either arm, whether you are standing cephalad or caudad to it. Dorsal flexion (see Fig. 4) brings the supraspinatus facet under the incision. Notice Figures 5 and 6, and observe that the supra- and infraspinatus partially overly one another. In fact, throughout the book I have reiterated that complete ruptures of the supraspinatus are very frequently accompanied by involvement of the infraspinatus tendon. This is important to remember when you are suturing the rent in the base of the bursa.

You may be assumed, now, to have looked over the records of your clinic and to have found and examined a number of long-standing cases of rupture of the supraspinatus. You have tried to see how well the eighteen criteria are verified in each. You have also made some dissections and think you know the anatomy of the normal shoulder, and are ready to operate on the first case which appears in your clinic. You will ask me how you are going to recognize it and not be misled by cases of less serious lesions, which will get well from nature's own methods. Perhaps you noticed in reading "The Shoulder" that I several times stated that I could only give the early symptoms by inference, because I had never seen a fresh case. Since that was written, over four years ago, I have had the good fortune to see the patient alluded to above, only twenty-four hours after the accident. The symptoms were about as typical as in the old cases, and were readily recognized. To my surprise, they were not quite so severe as in the old cases, nor was there any great degree of spasm of the protecting muscles as I had suspected might be the case. I have also seen several other patients only a few weeks after their accidents. Their symptoms were the same as in the old cases.

Fig. 4. Operative position. This is an important figure. The writer did not realize the advantage of placing the patient in this position until he had done many of these operations, which had been attempted with the patient either in recumbency or in a sitting posture strapped in a chair. The latter was a very satisfactory position after the strapping had been adjusted, if the assistants could be trained to work in this unusual attitude. However, the cases were too infrequent for any routine to be established. Until one operates on these cases one does not realize how elusive is the shoulder on the operating table, and, since the field is entirely controlled by the position of the humerus in relation to the scapula, how any minute change in the position of either the arm or the body is at once transmitted to the tiny wound. Gradually, I worked out this position, and find that it gives the best possible exposure, and enables one, if the assistant is attentive, to perform a very satisfactory suture through the very small incision.

You will ask what are the conditions likely to be confused with the immediate symptoms of complete rupture of the supraspinatus? I have half a mind to say—"only one important one"—and that is incomplete or partial rupture of the

tendon. But that would only be true after a good *x*-ray examination has proved negative. Without good radiographs two head of the bone in internal rotation, because the patient objects to having the arm rotated outwardly. The roent-

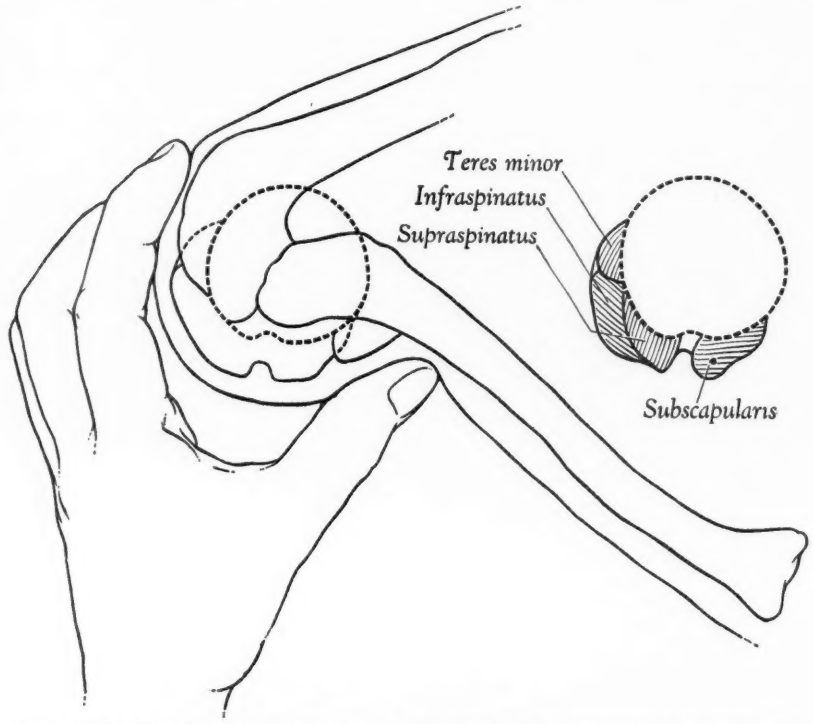

Fig. 5. The shoulder seen from above. The head of the humerus is completely protected from above and behind, and the contour in front is formed by the tuberosities. The inner contour on which the thumb naturally comes when the shoulder is grasped is formed by the tip of the coracoid. The forefinger sinks under the edge of the acromion. Notice that part of the facet for the infraspinatus lies in front of that of the supraspinatus, and compare with Figure 6. The position of the bicipital groove varies with the degree of rotation of the arm. Try this grasp on your own shoulder and rotate the humerus with the elbow flexed to a right angle. Bear in mind that the bicipital groove lies just outside of the median line of the upper arm in the anatomic position.

common conditions might readily be confusing, namely: cases of fracture of the greater tuberosity and certain cases of calcified deposits. You will notice the word "good" which applies not only to clear detail, but to a systematic method of taking the films so as to show in profile each of the facets of insertion of the four short rotators. To do this satisfactorily requires a considerable amount of study and practice on the part of the roentgenologist. I like the methods described by Blackett and Healey in the *American Journal of Roentgenology*. If radiographs are taken of an injured shoulder without special pains, they nearly always show the genologist must learn to try to take the head of the humerus and to disregard the position of the arm in relation to the body. It may be necessary to rotate the patient. The true anteroposterior view showing the supraspinatus facet will be taken when the path of the rays is parallel to the long axis of the ulna, the elbow being held in right angled flexion. The path of the rays should be on a level with the tuberosities, or better still if the tube is anterior, slightly above them; if it is posterior below them. If the shoulder is not very painful it is easy to get a lateral view by leaving tube, patient, and plate in the same position, and rotating the humerus

internally and abducting it about 45 degrees at the same time. This will place the arm akimbo and show the profile formative and oblique views will do almost as well, if one knows just how they were taken.

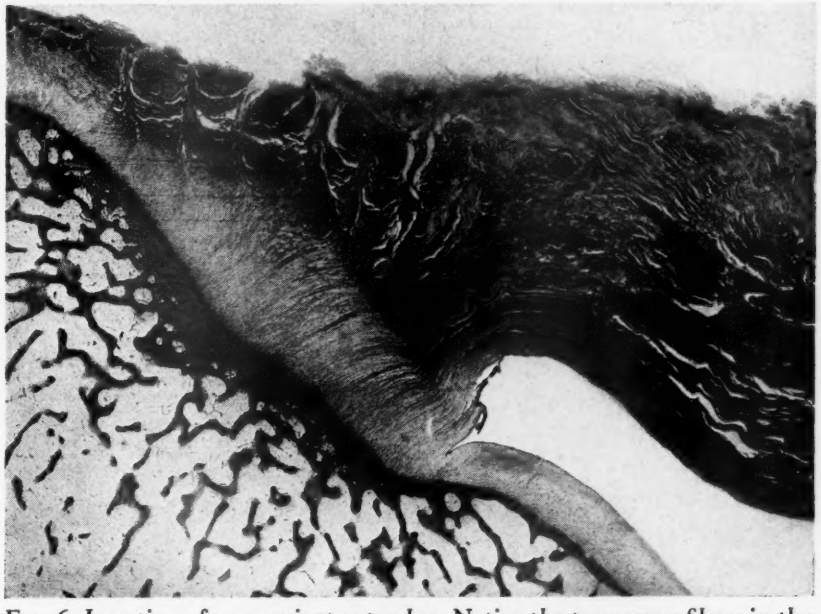

Fig. 6. Insertion of supraspinatus tendon. Notice the transverse fibers in the upper portion of the tendon. These are probably some of those of the infraspinatus. See in relation to this, Figure 5, which shows that the insertion of the infraspinatus overlaps that of the supraspinatus to some extent. Each of the other tendons also interlaces its fibers to some extent with its neighbor's tendons. Notice that even with the microscope no distinction can be made between capsule and tendon in this region. The synovial layer of cells beneath the tendon and the finger-like processes which attach the tendon to the facet cannot be seen without more magnification. Notice the dense bone on the surface of the facet. This is increased in density in cases of inflammation and is shown in the *x*-ray. Notice the palisade-like structure of the fibrocartilage which often remains on the bone when the tendon is ruptured. Refer to page 89 of "The Shoulder" for a description of the usual points of rupture. Notice how close to the rim of the articular cartilage the fibers are attached and that a few of them in this specimen have given away at the very edge. Notice the lamellated structure of the tendon. It is crinkly because it was detached from the scapula before fixation.

of both subscapularis and teres facets. To get the infraspinatus facet requires the anteroposterior view when the humerus is rotated about 15 degrees inwardly and the tube is raised slightly above the level of the tuberosities, if the plate is posterior; or slightly below them if the plate is anterior.

If a patient has a really sore shoulder and will not allow the arm to be moved, it is always possible to get a good anteroposterior view by rotating the patient until he points the forearm at the tube. A good lateral view is, however, difficult. Fortunately, the lateral view is less important.

The reason that the usual picture, taken with the patient on his back with the arm in the sling position, is unsatisfactory, is that the outline of the tuberosity overlies the outline of the articular head of the bone. In this position, fractures of the tuberosity, in which there is not much displacement, may escape notice entirely. Also, there may be small calcified deposits in the tendons which do not show because their shadows are overlaid by that of the head of the humerus or scapula. In the true anteroposterior view, taken parallel with the long axis of the flexed ulna, the outline of the tuberosity and that of

the sulcus (anatomic neck) appear sharply defined with no background of bone. This view almost always catches the line of cleavage of the fracture, but it is well to take two other views with the humerus rotated about 10 degrees to either side of this axis, using the flexed forearm as a guide. If there are deposits in both supra- and infraspinatus tendons, one of these views is likely to show both separately, although the deposits may be very close together. It is important to know how many deposits there are if you operate. Stereoscopic views may also be of help, although this method is a little disappointing in the shoulder compared to its value in other regions. The roentgenologist should be aware of the fact that in the true anteroposterior view the bicipital groove shows as a vertical line in the head of the bone. The degree of rotation of the head of the humerus may be fairly accurately estimated by the distance this line is shown to be from either side of the median line.

The presence of calcified deposits is not compatible with *complete* rupture of the supraspinatus tendon, which is a far more serious lesion.

Apart from the *x*-ray there are a few clinical points which help us to distinguish a recently fractured tuberosity from a rupture of the supraspinatus. As a rule the individuals are younger; up to 35 or 40 the tuberosity gives way more easily than does the tendon, when forcible elevation occurs. As a rule ecchymosis is seen after fracture of the tuberosity although it may not appear until several days after the injury and be far below the lesion, usually external and anterior to the lower part of the biceps. I have never seen it in uncomplicated rupture of the supraspinatus. Apparently the blood from the fractured surfaces gradually seeps down the bicipital groove which is cracked by the fracture. (See "The Shoulder," Fig. 62.) These lesions may also be distinguished fairly well by careful palpation. In cases of fracture there is a definite enlargement about the tuberosity owing to the hemorrhage about the fragment. There is no sulcus, as there always is in complete ruptures, and may be in partial ruptures. When the callus has formed there may be an eminence. In calcified deposits, at the place where the sulcus should be in complete ruptures, there is an eminence, and usually a very tender one. Calcified deposit cases seldom give a history of trauma of a degree adequate to rupture a tendon, but the cooked-up histories, which a patient invents to place his condition in the compensation class, are sometimes very puzzling. Moreover, I have seen a few cases in which I believe there was a bona fide combination of the two lesions; a symptomless deposit being made acute by an accident which ruptured those fibers of the tendon superficial to the deposit, precipitating an acute bursitis. The findings at operation seemed to prove this sequence of events. Yet we know that very trivial efforts may cause this discharge of the deposit from the tendon into the bursa.

The real difficulty in early diagnosis, when the *x*-ray is negative, is in deciding whether in a given case the rupture is extensive enough to need an operation. Where the symptoms and signs are definite and typical, and the sulcus is plainly palpable, there should be no doubt, but in cases of partial rupture each symptom may be milder, and each sign somewhat more doubtful, than those in the complete cases. Let us refer again to the eighteen criteria and take them up one by one, discussing them to see whether there may be help from any of them in determining the extent of the tear, and whether or not it extends through into the bursa.

1. In checking the lists of occupations in which these conditions occur, it is plain that we can get no help on this point.

2. The average age (page 124 in "The Shoulder") of complete cases was 55 and of partial cases 49. Since we believe that somewhere about and below 35 the tensile strength of the tuberosity is less than that

of the tendon, we might assume that at 49 the tendon would be relatively firmer than at 55. In other words, the younger the man the more likely would his injury be to prove a "partial" case, if he has not broken the tuberosity.

3. A "partial" tear would be more likely to have a clean history, for it is probable that once there has been a partial tear there is a greater likelihood of other injuries, but most patients with complete rupture allege that they have had no previous trouble.

4. The character of the accidents listed on pages 141–145 gives a fair idea of what may be considered an "adequate" injury in complete cases. We cannot assume that the degree of rupture would vary with the degree of violence. It is true in a general way, but the probable character of the tendon in feeble versus vigorous individuals would influence our judgment. We would expect either complete or incomplete lesions to come from the same kind of injury, but usually the complete cases give a very definite history of a severe fall. Occasionally, there is no such history and this is in favor of Myers' hypothesis.

5. Immediate, sharp, brief pain. I have no data to study in this connection. Both classes of cases usually allege that they had such a pain, but the complete case usually adds that *from that time* he could not raise his arm, while the partial case is often uncertain as to when he began to notice that he could not raise it. Usually he rests a little, then continues to work, and does not have much disability until the second or third day or even a week or two later. My impression is that this first pain is decidedly greater in the complete cases and that (6) the pain which comes on the night of the injury and continues for several nights is much greater in the complete cases. I am sure that (7) loss of power is more pronounced in the complete cases, although I cannot substantiate the statement with any statistics. In partial cases it is not a real loss of power; it is reflex inhibition of effort due to pain from making the tendon tense.

8. The *x*-ray is negative in both partial and complete cases—at least for several months.

9. Neither partial nor complete cases have restriction in the first few weeks, and it does not occur at all in complete cases, except in extreme degrees of motion, when it is probably due to the presence of fluid. (See page 151.) Partial cases, however, very frequently become restricted after a few weeks or months, and present typical "frozen shoulders," so that as time elapses it becomes easier and easier to distinguish the two conditions. Complete cases do not have adhesions. Let me warn you again and again to test for restriction of motion when the patient is stooping. When he stands, protective spasm of the muscles may lock his joint, but when he stoops gravity pulls the arm away from the shoulder and unlocks the spasm. (Fig. 7.)

10. Faulty scapulohumeral rhythm is present in both complete and incomplete cases from the first, but I think it is more pronounced the greater the extent of the tear. I have usually found at operation that the tear is greater in extent than I thought it would be, but I have always been conservative in advising operation—too conservative, I think, for exploration does not harm except to leave a little scar.

The examination of these cases is to me personally the most helpful thing in deciding the question of whether or not to operate, but one's reliance on this is only gained by experience. I have listed on page 169 all the errors that I had made in this diagnosis up to February 18, 1930, and I recall no instance when I have made the diagnosis of complete rupture and have failed to find one in the last eight years. Yet I have probably made the reverse error in some cases and failed to operate when I should have done so. I know that in at least one case this was true, for I postponed my diagnosis for

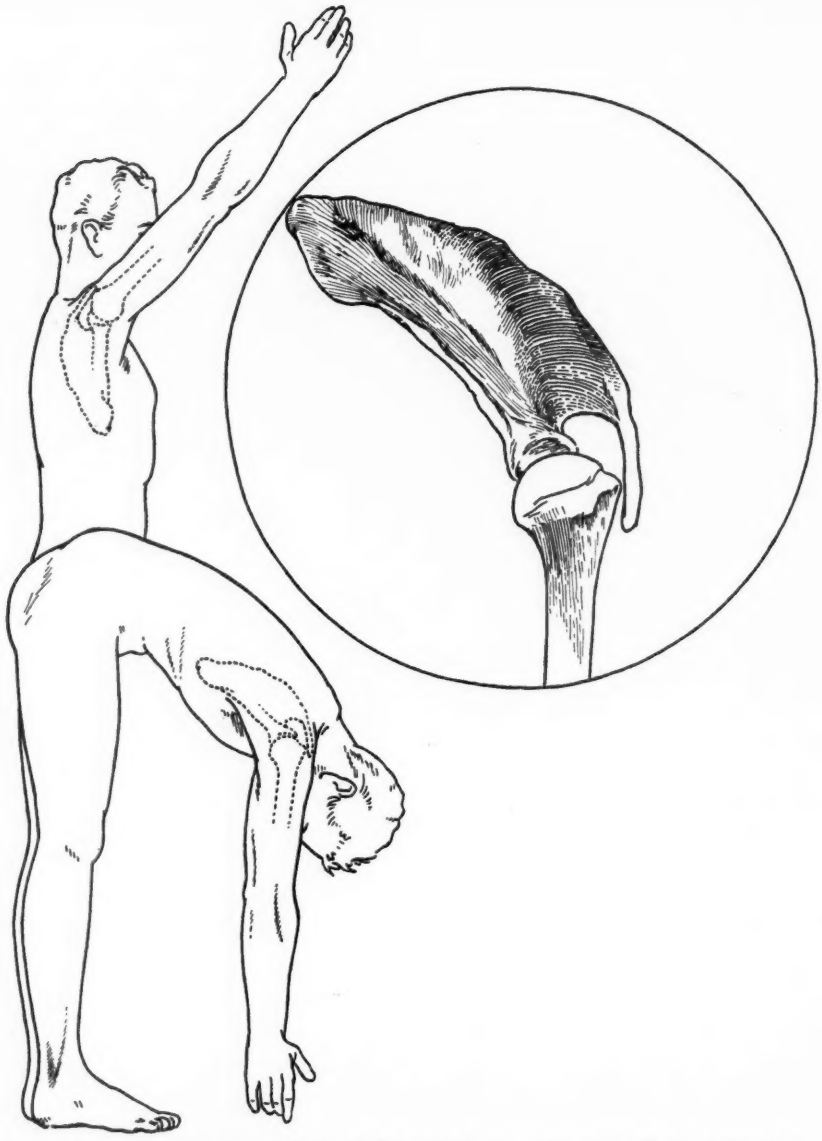

Fig. 7. Stooping exercises. The principle of the stooping exercises is that they permit the patient to abduct the arm by gravity and therefore no fulcrum is needed. If a patient elevates the arm in the standing position, the humerus obviously must obtain a fulcrum on either the glenoid or the acromion or both, while in the stooping position no fulcrum is necessary. In the standing position the little supraspinatus must pull the tuberosity firmly to keep the latter from touching the acromion. In the stooping posture it may remain relaxed.

Since the sore point in most lame shoulders is on the tuberosity at just the region where it tends to obtain a fulcrum on the acromion, it is highly important that the patient with a damaged supraspinatus should not delay his recovery by frequently raising his arms. The exquisite tenderness which arises in these cases is nature's defensive effort to warn the patient not to use the arm in this way. When the patient with the sore shoulder is in the stooping posture he can much more readily brush his hair, button the back of his collar or perform any other motions. He can, for instance, learn to put on his shirt and coat over the lame arm when stooping, and then, after he has risen to the erect position, to put on the other sleeve. When in the stooping position, either lateral or anteroposterior motions can be done with a pendulum-like movement without great muscular effort. In this position the humerus not only tends to avoid a fulcrum, but actually the weight of the arm helps to stretch the contracted tissue of the joint.

two days in a case which I saw with Dr. Henry Marble who had made the diagnosis. I waited for a negative *x*-ray before I would agree with him. He operated on the fourteenth day after the accident, found a typical complete rupture, and easily sutured the tendon accurately.

11–18. Examination deals with criteria 11 to 18 inclusive, and these may be considered together. Usually in complete cases, one has no doubt whatever as to the presence of each of these points. One can definitely place the finger tip in the sulcus (12) which is the rent in the tendon and feel the tuberosity (the eminence) to the outer side of it. Perhaps I have unduly accented the location as being at the supraspinatus insertion (14) for it is quite as well to say infraspinatus (see Fig. 6) since the two overlap and are both often involved. The jog (15) and soft crepitus (17) are readily felt and are so characteristic that even after noting them in only one case I believe they would be recognized a second time. They were just as definite in the case I saw twenty-four hours after the injury as in all the other cases. The complaint of tenderness (11) is so definite that patients usually describe it in the same words "You have got your finger right on it." The wince (16) is also very characteristic and occurs (18) as the irregularity caused by the sulcus and eminence passes under the acromion. Patients lean to the side as this occurs in order to diminish the pain. The maneuver is very characteristic. It is, of course, coincident with the jog and crepitus.

Thus the diagnosis of complete rupture is a very positive one, but that of incomplete rupture is not. One may be in doubt of a definite sulcus, the crepitus may be feeble, the eminence may only be a spurred tuberosity made more prominent by a puny tendon which does not round out the contour at the base of the bursa. One can only learn by experience to distinguish the sulcus made by the contrast between a spurred tuberosity and a puny senile tendon, from the sulcus caused by a rupture. For this reason one should seize every opportunity to examine the shoulders of patients with other injuries.

Always examine both shoulders. If the location of the tender spot is on the eminence, not at the depressed sulcus, and the patient lets the elevated arm down to his side without an obvious wince, it is unlikely that the rupture is complete. The doubtful character of the examination, combined with the presence of a considerable amount of power to hold the arm in the "scarecrow" position, pretty well rules out a complete rupture, although each of the criteria may be present in some degree. A patient with a complete rupture seldom can hold the arm at right angles with the body and withstand the slightest downward pressure on the hand. This is a very important test between complete and incomplete ruptures. This test is always very positive in complete cases, though dependent on the coöperation of the patient.

Later, perhaps in three weeks, atrophy of the spinati will become obvious, and persist for months whether the rent is complete or only a "rim" rent. You should always note the presence or absence of atrophy at your first examination. It is not present to any definite degree for two or three weeks. If it is present at first it is good evidence that the patient has had previous trouble of some kind with his shoulder.

You will ask what to do in case the diagnosis of incomplete rupture is made clinically or after an exploratory operation. No operation has yet been devised to reattach the fibers of the supraspinatus which may be torn under the base of the bursa. An attempt to do this might even prolong the convalescence. The presence of the intact base of the bursa furnishes a source of blood supply to the damaged tendon, and it is likely that it may heal satisfactorily.

In my opinion, there are two indications for treatment in the incomplete cases. One is to prevent atrophy of the muscles and the other is to prevent adhesions. The

patient should not work or take any strenuous exercises. The more he does so, the more his shoulder muscles will become spasmodic and hold his shoulder "frozen." Adhesions will then take place, and you will have a typical case of tendinitis, which may drag on for a year and perhaps become included in your second pile of histories. Whatever "frozen shoulders" you find in that pile will probably be instances of this sequence. The proper treatment would have been to make the patient stop work and let him do gentle stooping exercises (see Fig. 7) perhaps ten times each, and about three times a day. They will not start up spasm, but by putting the joint through its arc of motion a few times a day, keep adhesions from forming and the muscles from becoming atrophied. It takes very little exercise to keep muscles from becoming atrophied, but a few weeks' confinement in a sling or Velpeau will encourage atrophy. So will persistent protective spasm if the patient tries to work with a sore shoulder.

The reason we never get restriction of motion in the case of complete ruptures is, I believe, because so much fluid forms in the joint that it pumps back and forward in and out of the bursa with the motions of the arm, and tends to dilate both bursa and joint. When one incises the bursa in these cases fluid nearly always gushes out of the incision as the arm is raised. If there is much fluid it restricts elevation a little and changes the contour of the shoulder when the arm is elevated. (See page 155.)

The various forms of physiotherapy are being used today throughout America in the treatment of practically all shoulder injuries. In the case of complete ruptures I feel sure no good is done. In the case of incomplete ruptures I doubt if the convalescence is shortened, but it may be, and perhaps it is well to use the various "modalities" to entertain and encourage the patient. The relief of pain gained by each treatment is not great, for both in complete and incomplete cases the pain is bearable, if the patient rests and does not attempt to use the arm much. Vigorous exercises, forced manipulations and confinement in slings or bandages I believe to be actually harmful. Judicious massage —a hard thing to obtain in quantity— may be helpful in keeping up the circulation of the tissues.

Some of the readers of this article may be among those who have really studied "The Shoulder" and will say "All this I have read before. There is nothing new in it. Has he not learned anything more since the book appeared in 1934—nearly five years ago? He invited criticism and someone must have accepted the challenge either in surgical literature or by personal letters? What additions have been made to general knowledge about the shoulder and its diseases since 1934, by other men?"

These questions may be answered in order. My personal experience during these five years has added little of real communicable value. It has as a whole been confirmatory of what I have previously stated, especially as I hear from other surgeons who have had similar experiences. I know of but one paragraph in the book which I should like to withdraw and that is the last one on page 502, in which is described a case which I then thought was a rupture of the deltoid muscle. I now think the case is one of intramuscular lipoma, but this has not been proved by operation. A symptomless tumor remains palpable without much change.

In little details, the methods of operation have been changed, especially the method of closure of the rent in the base of the bursa. I now incise the upper falciform edge in its center and carry the incision back under the acromion until I can feel the base of the coracoid process above the glenoid, using care not to get as far back as the suprascapular nerve. Then I am sure of the location of the upper edge of the subscapularis and of that of the supraspinatus. The long tendon of the biceps will be exposed between them. Next it is

well to excise the falciform edge of the rest of the rent back to sound tendon. This means paring off about ¼ inch from about ⅔ of the edge of the rent. The other third, formed by the tuberosity or a stub of tendon on it, usually has no falciform edge and often in old cases consists of the bare bone of the facets of the former insertions.

This bony portion should be refreshed and drill holes made outward to the base of the tuberosity. The frontispiece of "The Shoulder" shows this bare area well. The edges to be sutured are now in the form of a long equilateral triangle whose short base is the tuberosity and whose apex is over the origin of the long head of the biceps, above the glenoid. Formerly I used to try to pull the apex down to the base, but since I have now seen several cases of fresh ruptures, I have learned that the apex does not belong at the base, because the combined retraction-pull of the supra- and infraspinatus is not upward but outward, i.e., away from the edge of the subscapularis. The latter is often uninjured and merely somewhat retracted inwardly—perhaps just enough to expose the long biceps tendon. It is possible to pull the edge of the subscapularis outward to meet the edge of the supraspinatus, much more easily than I used to think. I believe that by using this method, patients whom I used to think beyond repair can have successful suture. (Plate I.)

If one keeps in mind that the muscle bellies of three of these muscles are separated by bone for most of their extent, it is easier to understand how a tear in their conjoined tendons should be sutured. The edge of the scapula above the glenoid, i.e., the base of the coracoid process, separates the subscapularis from the supraspinatus, and the spine of the scapula separates the supraspinatus from the infraspinatus. This applies only to the bellies of these muscles, for soon after they have left these bony barriers, they become more or less tendinous. All of the tendons fuse into what I call the musculotendinous cuff, uniting with the capsule beneath them and at the same time with the base of the subacromial bursa. (Fig. 8.) All these structures then become inserted into the bony sulcus called the anatomic neck and the fused tendons of the subscapularis and supraspinatus become part of the roof of the bicipital groove, uniting with the small intertubercular ligament. Part of the superficial fibers of all the tendons pass down into the periosteum beneath the base of the bursa. Longitudinal sections taken through the insertion show that even with the microscope the welding is so complete that the individual layers cannot be made out. (See Fig. 6.) Their terminal portions for about an inch cannot be separated without force or sharp dissection.

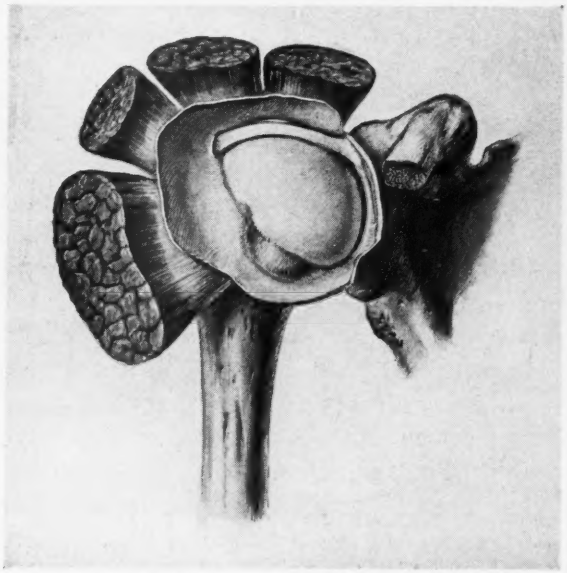

Fig. 8. The inside of the joint is shown from the anterior aspect especially to indicate that there is no sulcus in the upper half of the anatomic neck and that in the lower half of the joint the insertion of the capsule is some distance from the articular rim. Here the bone is covered only by a thin layer of fibrous tissue and synovia. The capsule has been cut just proximal to the line where it becomes welded into the musculotendinous cuff. The author wishes to emphasize the fact that in autopsy specimens one frequently finds bare bone between the attachment of the tendons and the articular cartilage and that such conditions are the end results of unrepaired ruptures of the tendon fibers.

Skinner has made an important contribution to the anatomy of this region

by showing that in children the muscles can be separated almost down to the tuberosity, and that as age advances, the distal portions become more and more tendinous and more intimately welded together.

It was due to seeing a few fresh cases in which the parts could be easily replaced, that I began to realize that it was unwise to try to pull the apex of the triangle to the middle of the base. In these cases falciform edges had not yet been formed and the margins of the rent were blunt and could be accurately fitted together. This taught me to suture the triangle as in the diagram (Plate 1) instead of in the manner shown in the diagram on page 240 of "The Shoulder."

The next question to answer is whether my critics have set me right, either publicly or privately. The criticism I most value was from Mr. Johnston, Professor of Anatomy at Guy's Hospital Medical School in London. He has given, in the *British Journal of Surgery*, an excellent interpretation of what I call the "pivotal paradox" and I heartily recommend his article to the reader, although Professor Johnston says that my proof "cannot be accepted." I would like to have you read that article because I feel that no man should undertake to treat patients with shoulder lesions until he knows the normal motions of the shoulder well enough to at least understand Professor Johnston's article, (this in spite of the fact that I am still convinced that my "pivotal paradox" *must* be accepted). What Professor Johnston forgets is that in my proof of the paradox, it is the descent of the arm of which I speak. What I say is true, but I do not explain the real trick, which is the fact that 90 degrees rotation will be necessary to return the arm to the anatomic position at the ends of both of the maneuvers. Johnston evidently ignores the anatomic position. So I beg the reader to understand his article if he wishes to be informed about the normal motions of the human shoulder, although I will warn him that he cannot expect to understand the article in a few readings. Johnston sees the truth, but his way of ignoring the anatomic position as a starting point has made him fail to see the paradox.

L. E. Snodgrass of Philadelphia has also criticized the paradox in long personal letters. He agrees neither with me nor with Johnston, but has himself written a good article on shoulder motions, although he had not seen my book when he published it. Another excellent article on the motions of the shoulder has been published by McGregor of Johannesburg, South Africa.

Those who teach anatomy to medical students should read these articles and decide how to teach the subject to their students, for future doctors must know more about the shoulder than is generally known today. Snodgrass' errors are mainly due, I think, to his failure to appreciate how much more movable the shoulder joint may be in the living person than in the stiffened cadaver of the dissecting room.

Johnston has done a real service in suggesting that anatomists should teach that motions of the scapulohumeral joint should be described in relation to the plane of the scapula. I should define this as a plane passing through three points, although this is not exactly Johnston's definition, viz.: (1) the tip of the acromion; (2) the tip of the inferior angle of the scapula; (3) a point on the vertebral border of the scapula adjacent to the small triangular surface at the inner end of the spine of the scapula. This plane cuts the glenoid in halves through its long diameter. By using this plane the possible positions which the humerus may take in relation to the shoulder blade may be more definitely described, and therefore their motions on one another, more satisfactorily discussed. For instance, Snodgrass and I frankly differ on the possible degrees of rotation of the humerus at the scapulohumeral joint when the arm is at the side. He thinks the normal less than 90 degrees, while I consider that it is 180 in youth and that the number of degrees slowly

diminishes with age, but always remains above 90 in a normal adult. In such a discussion we must admit the fact, of course, that the amount varies considerably in individuals but not so greatly as it does in living persons versus dissecting room subjects.

If I ever write a third edition to "The Shoulder" (which I do not plan to do) I should certainly use much of Johnston's article, because the reports of physicians on the degree of arcs of motion in shoulder cases are as a rule almost ridiculous. They do not distinguish the extent of motion in the scapulohumeral joint from that in the clavicular joints. In fact, at present, few readers would be qualified to understand such a distinction. There is little agreement even in the use of terms to apply to the motions of this joint. (See pages 46 and 47.) Such descriptions are, to be sure, more difficult, but by using Johnston's suggestion of referring to the plane of the scapula we might discuss the normal motions far more intelligently.

Another critic, many of whose views I commend, but with whom I frankly differ on others, is Dr. Arthur Meyer, Professor of Anatomy at Leland Stanford, who has published many excellent articles on the effect of friction in living tissues. I have alluded to some of them on pages 184–242–497 and 498 in "The Shoulder." I have recently visited Dr. Meyer and have seen many of the specimens on which he bases his argument for the attrition theory. We are satisfied to continue to differ as to the probable etiology of most of his shoulder specimens. The very lesions which I am confident are partially healed ruptures of the supraspinatus are used by him to support his attrition theory. I regard the falciform edges as new formed tissue which is thrown out to make the blunt edges of the rupture less of an impediment to elevation of the arm, while he is equally sure that the tendon was worn thin and the falciform edges are remnants of the original tendons. (Codman.[1])

My chief argument is the fact that I have seen these ruptures in all stages and that soon after the injury the falciform edges are not present and the blunt edges can be readily united. Dr. Meyer's chief argument is the finding of similar evidences of attrition in other anatomic situations. There are also many other points favoring one side or the other, too numerous to be discussed here.

This question will not be decided by men of Dr. Meyer's and my generation. There is much evidence to collect and analyze, for as yet the clinic and the anatomic laboratory do not often deal with the same material. When patients with histories of obscure shoulder lesions can be followed to the dissecting room, the answer will probably become plain. Such a piece of research could be done at any large hospital for chronic cases.

The question as to what surgical literature has added to our knowledge about injuries of the shoulder can be easily answered, for I need only refer to an article written by Dr. L. K. Ferguson of Philadelphia.[6] The literature has been most carefully and judiciously reviewed by a man thoroughly competent to do the work. The chief points of advance have been in the way of treatment, especially in the use of novocaine and sterile salt solution to allay symptoms, and in some cases to remove calcified deposits. The most important of these new methods of treatment seems to me that of Smith-Peterson, who inserts two needles into the deposit, syringes salt solution into the smaller one and washes the particles of calcium out through the larger one. Dr. Smith-Peterson has not contributed an article himself, but two of his former assistants, Drs. Norton and Patterson have done so, the former using material from the Massachusetts General Hospital Clinic in Boston and the latter from the Presbyterian Hospital Clinic in New York.

I have had such complete success from the trivial operation described in my book (page 226) that I continue to use it, but I am convinced that this needling method is of value in some cases, though not as certain as the operative method. It

has the advantage that the patient may remain ambulatory, while if an operation is done the patient should be hospitalized for a few days at least. Some people would consent to needling rather than to have an "operation with a knife," simple as that it may be!

Only one article has appeared which, it seems to me, tends, perhaps, to make us take a step backward. I allude to an article by V. L. Schrager of Chicago (*Surg., Gynec. & Obst.*, April 1938). The author states that he is describing a new clinical entity, tenosynovitis of the biceps tendon, which he believes is the most common cause of sore shoulders. In "The Shoulder" (page 146) under the heading "Tenosynovitis of the Biceps," is the following: "When I was in the medical school, I used to hear this diagnosis made . . . There may be such a lesion as tenosynovitis of the biceps tendon, but I have never demonstrated it either clinically at operation, or at autopsy except in conjunction with ruptures of the supraspinatus."

I feel no impulse now to amplify any statements made in the paragraph from which these sentences were quoted, even though I have read Dr. Schrager's article. I feel that we have left the lesion in question behind and that it seldom need be considered in diagnosis. Partial ruptures or shredding of the biceps, so well described by Gilcreest[8] is undoubtedly a real diagnosis on which intelligent treatment may be based. However, Dr. Schrager may be right, and his ideas are to some extent in accord with Meyer's belief, founded on years of work, that attrition of the biceps tendon occurs from overuse, apart from any acute trauma or exposure following rupture of the supraspinatus. It would seem logical to assume that in this condition synovitis would appear at an early stage. I do not wish to influence others against Dr. Schrager's and Dr. Meyer's views, but merely to state that after years of study of the shoulder I cannot yet accept them. I think enough of these authors to recommend that you study their arguments as well as mine, although I regard theirs as misleading. It is hard to observe carefully and record facts accurately, but it is even more difficult to interpret them correctly. In this case the facts are fairly clear, but there are two diametrically opposed explanations of causes. Yet nature at times permits two different causes to bring about similar effects, and even combines causes to produce effects. Dr. Meyer would not deny that tendons worn thin by attrition might break. Nor would I deny that the more worn or senile the tendon, the easier it would be to break.

I may be mistaken in feeling so strongly that if we could only operate on these patients soon after their injuries, we should save them much pain and many working hours. I have often been mistaken in my opinions; undoubtedly there are many errors in "The Shoulder." Please point them out publicly if you are sure of your ground; and if not, just write to me about them. Perhaps I can give you more evidence now, for there are many minor additions and changes which could be made in the book today.

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COMPOUND FRACTURES OF THE ELBOW JOINT IN ADULTS*

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THE increasing frequency of automobile accidents has produced a great number of bad compound comminuted fractures of the elbow joint. A large percentage of these are of the left elbow, due to the custom of driving with the elbow protruding from the car window. As the bodies of the cars are wider, when an accident occurs the arm is exposed to terrific violence.

A discussion of compound fractures of the elbow must be opened primarily with a full discussion of a method of treatment of compound fractures that has proved satisfactory, not only in the elbow, but in other regions. This method we have used successfully with 91 per cent clean cases, irrespective of the multiplicity of the injuries or the extent of the compounding. Where there is no great destruction of tissue or lacerated or incised wounds, we have nearly 100 per cent clean wounds. The method is as follows:

1. Cover the wound with sterile gauze.
2. Cleanse surrounding area and shave any hair present.
3. Wash and cleanse wound and edges of wound with soap, water and normal salt solution.
4. Debride all avascular tissues. Cut away dirty edges of wound with scissors or knife. If separate spicules of bone are dirty and detached they must be removed. If dirt is ground into one of the bone fragments the dirt may be removed from this bone fragment with a chisel or a rongeur. Irrigate freely with normal salt solution.
5. The wounds are sutured loosely, the sutures being stitches from 1″ to 1¼″ apart. Dry dressings are applied and changed every six hours during the first two days in order to obtain the maximum capillary drainage and to prevent any fermentation. When the marked extravasation of serum decreases, the dressings may be left in place until the removal of the stitches. This method, in our hands, has been far more satisfactory than the use of Dakin's or any other group of antiseptics.

The reasons why wounds treated in this manner remain clean are:

1. The skin edges are loosely coapted and this permits the subsequent oozing of blood to carry away froeign bodies that have escaped the observation of the surgeon.
2. The serum that is thrown out later by every wound tends to dissolve and force out any excess blood clot, nourish the injured tissues, and carry away foreign bodies, as well as destroy dead tissues.
3. The dry dressings afford a maximum capillarity, so that all serum and blood that has been extruded may be rapidly absorbed; and the six-hour change of these dressings is sufficient to prevent fermentation or growth of bacteria in the extruded secretions.

* From the Fracture Ward of the Cook County Hospital, Chicago.

4. These wounds are never washed with an antiseptic, either at the time the patient enters the hospital or after the loose closure of the wound.

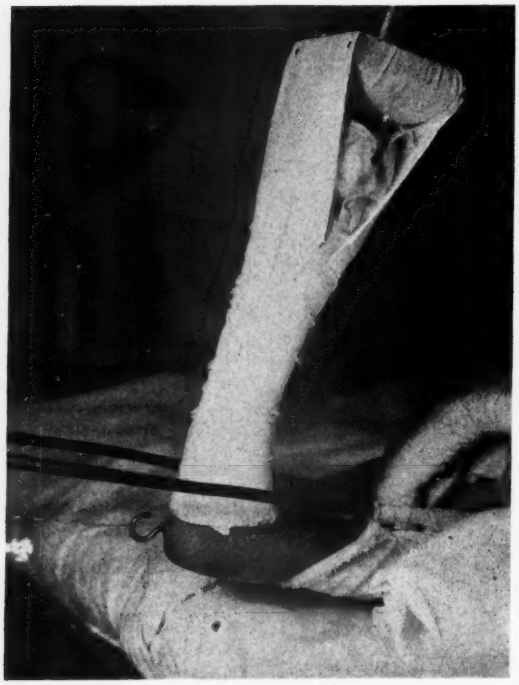

Fig. 1. Shows hook screwed into flat area of olecranon with traction attached. Forearm and hand supported with Buck's extension. Thomas splint extends to saw horse, which supports the outer end and carries weight of extension over a pulley.

5. The utilization of this washing process by blood and serum should be used more commonly, because it is an age-old protective function of the human body.

In all extensive compound fractures around the elbow joint, or anywhere else, antitoxin for gas and tetanus should be given in curative doses when the patient enters the hospital. We are convinced that the prophylactic dosage as is customarily given at the present time is inadequate. If a large dosage—20,000 units of polyvalent gas antitoxin and 5,000 units of tetanus antitoxin—is given early, there will rarely be an infection with either of these organisms.

In those cases of compound fracture around the elbow, admitted late with gas infection present, large dosages—150,000 to 200,000 units—of polyvalent gas antitoxin are given early and the wound is enlarged until it is possible to make a free débridement of all gas infected tissue. The surgeon must not hestitate to cut away all involved muscle and subcutaneous tissue and allow the wound to remain open, without sutures. Potassium permanganate crystals are then placed in the wound and hydrogen peroxide may also be used locally. Postoperatively, the potassium permanganate solution, 1:3000, is used and alternated every six hours with hydrogen peroxide 1:4. If one desires, a few perforated tubes may be laid on the tissues and every few hours some of the solution may be run through the system to the wounded surface. It is not imperative, however, to put tubes in the wound, as they can be irrigated with a hand syringe at intervals just as effectively, without the danger of the tube's causing a pressure necrosis in the tissues to which they are adjacent.

The dressings must be changed frequently. A heat cradle is placed over the wound, keeping the temperature at 100 degrees, which appears to be most beneficial for the circulation. This régime is carried out until the clinical picture shows a drop in temperature and improvement of the local lesion. We have never been forced to amputate an arm from any infection—and for one of us this covers an experience extending over thirty-six years. With the lower extremity, where there is lowered resistance, this statement does not hold true. Extensive gas infections extending up to the abdominal wall from an infection in the lower extremity, when treated in this manner, have ceased to spread.

We are positive that the *x*-ray treatment of gas bacillus infection has little, if any, value. In eight cases of proved gas infection that were treated with *x*-ray without surgery or antitoxin, there were eight deaths.

One author claimed that surgery was harmful in these cases. We consider this a dangerous statement. In our opinion

it is absurd to conclude that *x*-ray will be of any value in the treatment of a gas infection in tissues, when *x*-ray primarily will destroy tissues and no amount of it will serve to disturb the growth and reproduction of gas bacilli when it is applied to cultures. We can see no reason, in view of these well-known facts, for even considering *x*-ray in the treatment of these cases.

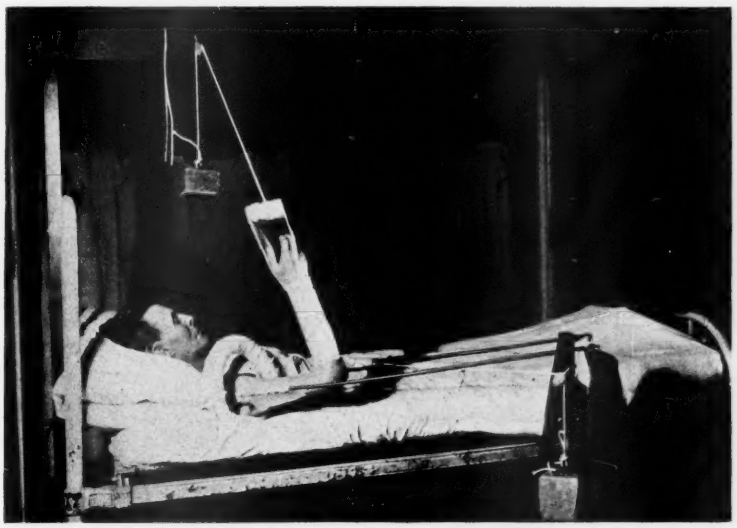

Fig. 2. Similar to Figure 1. Shows a longitudinal traction. Thomas splint. Saw horse support and pulley traction attached to hook screw.

In compound comminuted fractures of the lower portion of the humerus, in which we may have any number of fractures, it is our custom to insert an ordinary hook screw. In adults an incision is made over the flat portion of the ulna, which is about 1½ inches from the proximal end of the olecranon, and opposite the coronoid process. A hole is drilled into this flat surface with a drill 1/64 inch smaller than the threaded portion. Then the hook is screwed in firm and strong. A 5 to 10 pound weight (Fig. 1) varying with the size of the individual, is attached to this screw for traction, and the forearm is supported with either a Buck's extension (Fig. 2) or, if

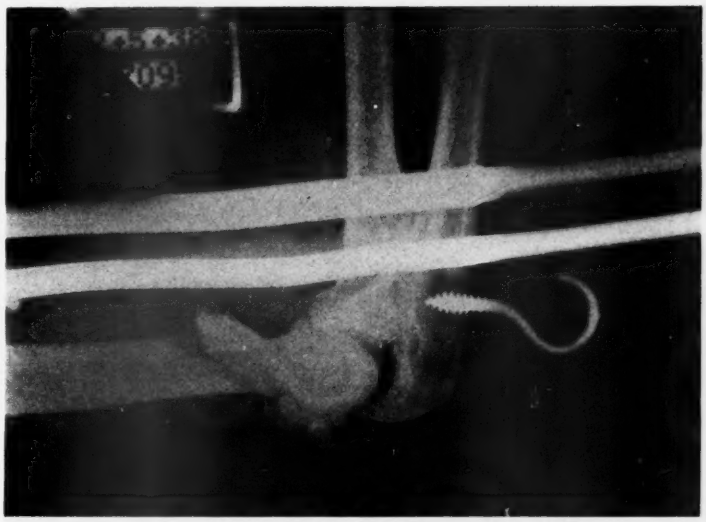

Fig. 3. *X*-ray showing screw hook in flat surface of ulna, opposite the coronoid.

there is a compound fracture present in the forearm, we may also use a Kirschner wire either through the metacarpal bones, fingers may be continued throughout the treatment.

Once in a while, with an extensive

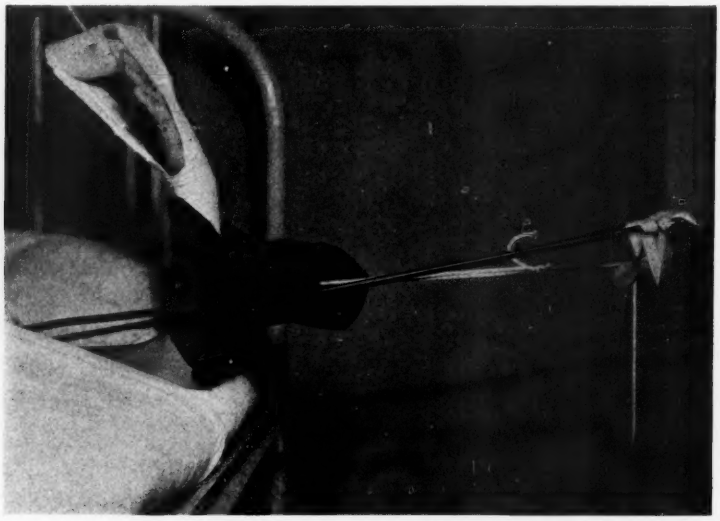

Fig. 4. Lateral traction by means of a piece of saddler's felt. To be used when ulna is so fragmented or compounded that a foreign body would not be indicated.

or through the distal end of the radius and ulna. If the arm must be extended along the side it can be accomplished by the traction as shown in Figure 3.

With a compound comminuted dislocation of the elbow, it has been our custom to reduce this elbow, flex it slightly and apply a moulded splint that extends from the shoulder down and around the arm, in such a manner as to avoid a point of pressure upon the open wound. By that we mean, if the compound is on the outer side of the elbow, the plaster splint, which is first moulded out upon a table, is placed over the shoulder cap, twisted down around and behind the humerus to the inner side of the arm and forearm, down to the mid-palmar. It is bandaged in place and, while it is still soft, any slight displacement or dislocation of the fragments can be reduced again and then held in the position required. It is quite obvious, if there is a great lacerated wound on the inner side of the arm and forearm, that the cast must be carried to the outer side of the arm and end at the metacarpophalangeal articulation, so that if possible the motion of the comminuted fracture over the ulnar area, where it is contraindicated to employ a screw or wire of any type, in order to obtain traction we use a well padded band over the flexed forearm, adjacent to the humerus. The flexed forearm is suspended with moleskin traction and weights are applied to the saddlers' felt to the forearm, in order to get longitudinal traction on the humerus. This must be carefully watched so as not to interfere with the circulation or the nerve supply of the muscles distal to the extension. This method is not as efficient as skeletal traction. (Fig. 4.) The arm is removed from this form of traction at the end of three weeks; as a rule by this time the reduction and callus formation is adequate to place the arm in plaster. The optimum position is right angle flexion of the elbow with complete supination of the forearm.

The use of these spiral plaster casts is imperative in many cases where no form of skeletal traction can be used. But it must be remembered in the use of plaster that no pressure can be made over wounded tissues, as pressure interferes with blood

supply and the life of the tissues. We have rarely used the cast around the upper arm and shoulder with U-shaped iron supports attached to a forearm cast. In our hands this apparatus seldom serves as a good support to the arm and forearm in these compound injuries.

In compound fractures of the radial head, combined with a compound olecranon fracture, there is usually an associated dislocation of the radial head and the proximal portion of the distal ulnar fragment upward, with retraction backward and upward of the olecranon process. In these cases, after careful débridement and the removal of only those fragments of the radial head that are completely separated, we drill the proximal portion of the distal ulnar fragment, pass a silk suture through it, then around through the triceps tendon over the proximal fragment, and pull those fragments into apposition. (Fig. 5.)

We are somewhat opposed to the free removal of the radial head in these fractures, because the radius constitutes a marked support of the ulna, when the distal radio-ulnar joint has remained intact, and will help avoid the marked curvation which so frequently occurs in this type of injury.

Where the lower end of the humerus, as well as the ulna and radius, is fractured and comminuted, we have not resorted to the use of metal plates, although occasionally we have transfixed a T-shaped fracture of the humeral condyles with a long chrome plated steel screw.

If one wishes to use a screw through the detached olecranon process, it must be remembered that this screw must be directed upward and forward toward the coronoid, in order to hold the fragments firm and strong. This method has been given careful study by Dr. William B. Carrell, of Dallas, Texas, with excellent results. However, in our clinic, we have found that any metal substance used in compound fractures has served to increase the frequency of infections. Silk or catgut is much better tolerated.

It would be well to mention here that if the radial head has been fractured with a dislocation and there has been an associated rupture of the radio-ulnar joint at the distal end, the removal of the radial head permits a radial angulation of the hand and a downward protrusion of the ulnar styloid, in such a manner that it impinges upon the carpal bones, causing pain and discomfort of such a severe type that it interferes seriously with the function of the hand. In several cases in which this has been done it has been necessary to remove the ulnar styloid before a useful hand could be obtained.

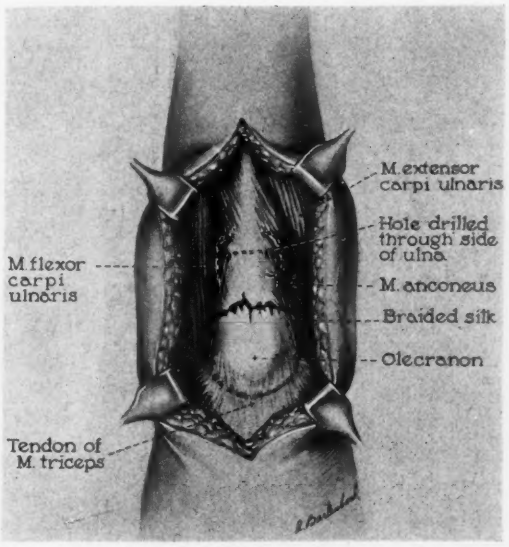

Fig. 5. Method of repair of olecranon fracture, compound or simple. Can be used to great advantage with a comminuted fracture of the ulna, as the silk circlage will hold multiple fragments in apposition.

It is well to mention here the slight displacements of some overriding in these compound comminuted fractures. In our opinion the slight deformities shown in the *x*-ray have been vastly overemphasized and should cause no change in the fixation or treatment. Of course, if there is a gross deformity, readjustment and a correct alignment must be made. But most of the slight misalignments shown by the *x*-ray can be let alone with perfect safety and with the assurance that a good arm will be obtained.

In relation to the movements of the elbow and the best methods by which motion should be obtained, it is well to caution very strongly against any forced motion. Forced motion around the elbow joint under anesthesia has served to cause us more disability than any other procedure and we have long since discontinued using it as myositis ossificans may result. It is best to begin motion, both active and passive, early. This can be done with the hook extension in place. And, as soon as the traction has been removed and the wound healed, the combination treatment of light, heat and gentle massage can be instituted.

Rarely does one obtain a maximum amount of motion in one of these compound comminuted elbow injuries before a year has elapsed, and we are of the opinion that it is far simpler and safer to open this elbow joint carefully, free the firm adhesions, remove any little exostoses that may be present and, in that manner, obtain the necessary increase of the elbow joint motion.

CONCLUSIONS

1. Under our present management of compound fractures we have 91 per cent clean wounds, regardless of extent of injury.

2. The use of the hook screw in the olecranon has saved many a mangled arm, with restoration of a functional elbow.

3. The screw has innumerable advantages over the use of the Kirschner wire, the most important being that the ulnar nerve cannot be injured, and the procedure is far simpler and more stable, since there is not the slippage which is so frequently present when the wire is used.

4. Avoid all force in the mobilizing of injured elbow joints.

5. Do not be greatly concerned about slight malpositions.

INJURIES TO THE CARPAL BONES

WITH PARTICULAR REFERENCE TO THE CARPAL SCAPHOID

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MUCH has been written in recent literature concerning fractures of the navicular or scaphoid bone, but apparently there is room for more emphasis on the care of this injury.

In a review of the literature it may be observed that the opinions on treatment vary greatly. We can find references to treatment extending from brief periods of three or four weeks to six months. These methods are far from standardized, and consequently there is a great variance in the end results obtained. A thorough knowledge of the functional importance of this bone in conjunction with a fundamental understanding of its anatomy and physiology is essential if we are to regard its injuries with the respect it deserves. Improper treatment will result in prolonged convalescence, and frequently a certain amount of permanent disability.

On many occasions the "typical" fracture involves not only the usual injuries of the radius and ulna, but also on careful examination, a complicating fracture of the navicular bone may be found. These cases treated in the usual manner for Colles fracture are certain to have a prolonged disability, and an ununited fracture may result. Such instances are not infrequent.

In order to thoroughly understand the need of the recommended treatment of these fractures, one should carefully consider the anatomy of the carpal navicular bone. The anatomy, with particular reference to the bony structure and blood supply, has been well studied and recorded by B. E. Obletz and B. M. Halbstein (Nonunion of fractures of the carpal navicular, *J. Bone & Joint Surg.*, 20: 424, 1938). In this monograph they point out the fact that in 13 per cent of the navicular bones studied there was no apparent blood supply proximal to the constricted midportion or "waist"; that in 20 per cent there was a single small arterial foramen at the waist or proximal to it; and in 67 per cent there were two or more foramina proximal to the waist. Therefore, it may be seen that fractures through the constricted portion cause a severe interference of normal blood supply to the proximal fragment, and in at least 13 per cent of the cases a total interception in the nutrition of this part.

The vast majority of fractures are at the narrow midportion. Since the navicular carpus is interposed between the greater and lesser multiangular carpi and the distal articular surface of the radius, it is probable that it becomes compressed between these surfaces when force is applied to the hand while held in dorsiflexion and radial deviation. Key and Conwell (Fractures, Dislocations and Sprains, page 672, Mosby, 1934) rightly say that it is impossible to state the exact mechanism of the injury because of the patient's inability to give an accurate account of his position at the moment of impact. One point in the treatment must be borne in mind because of the anatomy of the wrist, namely, that, in order to obtain complete immobilization of a fractured navicular bone, the proximal phalanx of the thumb must be fixed securely.

As already stated, most fractures are through the constricted portion. Usually there is little or no displacement and the fracture is simple in type. Occasionally there is comminution with more or less degree of displacement. Frequently the injury is associated with dislocation of the

semilunar bone due to severe force and an extrusion of the bone through the volar ligaments. Complicating fractures of the radius and ulna are not infrequent, the radial fracture lines usually extending through the articular surface.

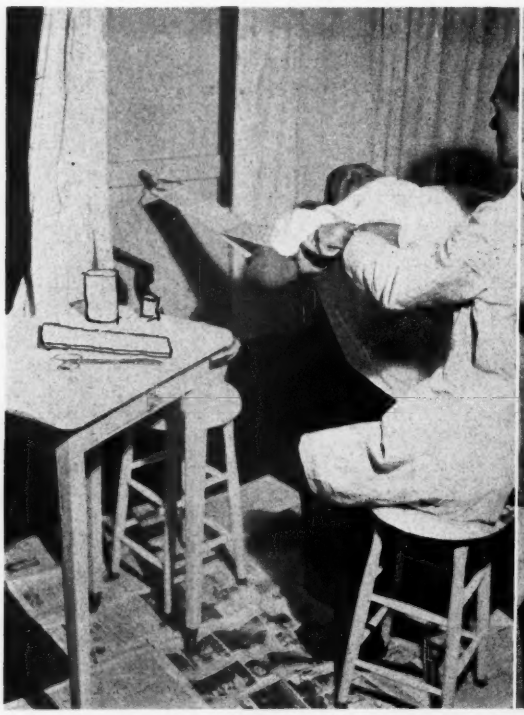

Fig. 1. Step I, showing materials, position of patient and assistant.

Fig. 2. Step II, gauze bandage about to be wrapped around arm and posterior mould.

Diagnosis of fracture of the carpal navicular bone is overlooked too often. Usually the condition is at first considered in the category of a simple sprain of the wrist. There is a history of adequate trauma. The symptoms are persistent pain most noticeable on pressure over the navicular region and in the anatomic "snuffbox," limitation of both flexion and extension with increase of pain on radial deviation of the hand, and persistent swelling.

Careful *x*-ray studies will confirm the diagnosis. Many fractures of this bone are overlooked because frequently the fracture line is hardly discernible, and, in case of doubt, the use of a magnifying glass in the study of the film may disclose the evidence of pathology. Views should be in the anteroposterior and lateral planes, and we now routinely make film studies of all suspicious wrist injuries in the oblique anteroposterior plane; that is, with the *x*-ray passed through the wrist at a 45 degree angle to the carpal plane. Thus the carpal bones are seen to be somewhat separated and elongated, giving opportunity for more careful and exacting study. The foregoing technique has been emphasized by Lorenz Bohler in "Treatment of Fractures" and we have found it exceedingly helpful.

Reduction of fractures of the navicular bone are usually quite simple. The occasional displacement of the extra-articular fracture of the tuberosity is accomplished by simple compression maintained by proper splinting. When complete volar or dorsal displacement of either a proximal or distal fragment is encountered, extension and manipulation by means of moving all of the carpi into alignment with the offset fragments will usually bring about a satisfactory position. We have not found it necessary to place the hand in radial deviation, and by experience we believe that the

best immobilization of the fragments is obtained by allowing the hand to remain in a slightly dorsiflexed position.

The most efficient means of maintaining immobility, and at the same time allowing the maximum amount of function, is the

Fig. 3. Step III, spiral plaster bandage holding all in place.

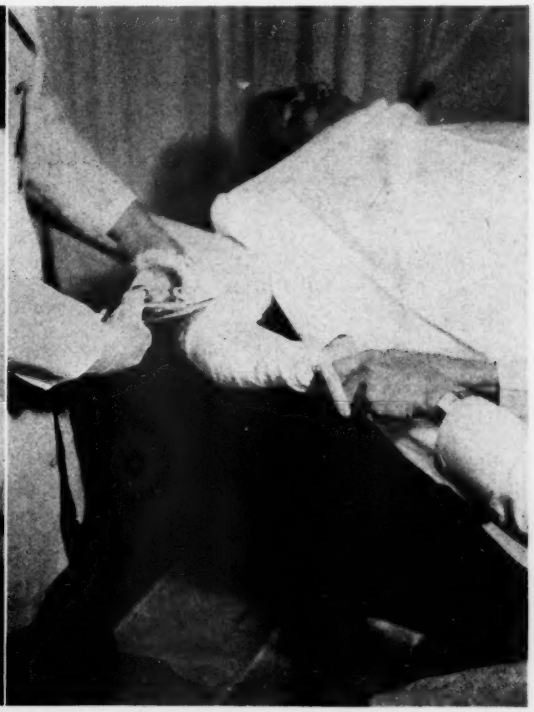

Fig. 4. Step IV, completed plaster being trimmed and held until hardened.

Reduction and splinting may be accomplished in the majority of cases without benefit of anesthesia in the nondisplaced group. In the nervous, painful, or displaced group, anesthesia is necessary. These latter types cannot be properly handled in the presence of muscle spasm, and painless handling is essential. Local anesthesia is preferable; we have employed it for over ten years without untoward effects. The earlier a fracture is treated, the greater the ease of anesthetization and reduction.

The method of immobilization which we believe to bring about the best results closely follows the Bohler technique and is herewith given in detail.

If local anesthesia is to be used, 1½ to 2 per cent novocaine solution is injected directly into the fracture site. The skin is always prepared with 7 per cent tincture of iodine or tincture of merthiolate 1:1000. The needle is handled with sterile instruments only, and a sterile technique is followed throughout the entire procedure.

use of the plaster of Paris splint. This is applied with ease and is most comfortable for the patient. After the wrist is made painless, the patient is placed in a supine position on an immovable table. A belt 4 inches wide is placed around the arm and fixed to the wall behind the head of the patient and at the level of the table. An assistant holds the injured hand by grasping the thumb in one hand and the index, middle, and ring fingers in the other hand. The fingers are held in such a manner as to prevent overlapping. Any manipulation necessary is done at this time. A mould of fast-drying plaster, 6 inches wide and of sufficient length to extend from the metacarpophalangeal junction to just below the elbow, is laid on the posterior surface of the forearm and carefully moulded to fit the contour of the area covered. Extension is not advisable. If the forearm is not supported, the wrist will fall into slight dorsiflexion, and this is the position of choice. A gauze bandage 2 inches wide is spiraled

about the mould and forearm. The entire arm is now thinly covered with a spiral of plaster, 4 inches wide. This spiral covers the forearm and also passes around the base of the thumb, not only immobilizing the proximal phalanx of the thumb, but also holding the hand snugly to the slightly dorsiflexed posterior mould. With the above splint we obtain the maximum amount of immobility due to the entire lack of padding and to the use of moulded plaster in the position and manner described.

Fractures of the navicular bone should be splinted for a minimum of nine weeks. Check-up *x*-rays taken at the end of this time must demonstrate firm osseous union; otherwise the cast must remain for a longer period. This long period of immobilization is necessary in order to allow sufficient time for the formation of new blood vessels in much the same manner as in intracapsular fractures of the femoral neck. With the aid of this form-fitting splint, moreover, the patient is quite able to carry on a large part of his usual duties.

The natural massage of functioning tendons and the improved circulation incident to exercise are highly beneficial.

FIG. 5. Demonstrating extension of hand and fingers and mobility of arm.

FIG. 6. Demonstrating flexion of fingers.

True enough, all patients will not coöperate to the extent desired, but the ones who have continued in active duty have found that their convalescence is shortened, and their mental and economic status is far better, as compared with the inactive patients.

One example of excellent coöperation was in the case of an ice delivery man who came every Saturday for a new splint because his week's work had completely worn out the splint applied at the previous visit. This patient had firm bony union at the end of nine weeks.

On removal of the splint it is gratifying to note the comparatively strong musculature and the lack of atrophy. The skin is healthy, and there is only slight stiffness. Physiotherapy in the form of a whirlpool bath and heat is occasionally given, but this is usually of short duration. We do not apply any supportive bandage on removal of the cast except in a few cases, and usu-

ally these need such a support for psychological reasons only.

Fractures of carpal bones other than the navicular are immobilized and treated in the same manner. Dislocations of the lunate bone, though comparatively infrequent, are sufficiently numerous to cause concern. In our series all dislocations of the lunate bone have been discovered and treated at the time of injury. The method of reduction has not been found difficult and has always been the same. With due respect to the various methods advocated, we have found that prolonged simple axial traction has accomplished excellent results. Adequate traction to supply sufficient space for replacement, prolonged until there is muscular relaxation, will bring gratifying results. Dislocation is practically always to the volar side, and by means of a steady traction of ten to fifteen minutes we have found that, with the aid of simple pressure over the dislocated bone, the flexor tendons force the lunate bone into position. Splinting is the same as already described, but immobilization is only necessary for a period of three to four weeks, providing active exercise has been continued during that period.

The treatment of old injuries of the navicular bone must be mentioned in any discussion concerning fractures of this bone. Obviously, early diagnosis and adequate treatment are the best prophylaxis for non-union. All suspicious cases should be *x*-rayed in the three advocated views, and, if there is persistent pain, a check-up series of plates should be made at the end of two to three weeks, at which time the fracture line will be easily discernible. Certain cases of non-union are bound to appear. Although removal of the fragments is frequently advised, we cannot bring ourselves to employ this disabling procedure. Bone drilling (six to eight drill holes through both fragments) with subsequent immobilization for ten to twelve weeks has met with excellent results.

We have studied a consecutive series of 669 of our fracture cases. We have found that 18½ per cent were fractures involving the wrist and, on elimination of the most common fractures, those of the phalanx, we find that 26.7 per cent involved the wrist bones. Of the wrist fractures 31.9 per cent were of the carpal bones. We found that a most frequent major fracture involves the wrist joint, and an appreciable proportion includes the carpals. Therefore, a thorough understanding of the diagnosis and treatment of these disabling injuries of the carpal bones is highly important.

Our average time from reduction to discharge of cases of a fractured navicular bone has been ten weeks. Most of our patients continue their usual duties, and all exercise to a far greater extent than we have observed during the use of any other method. We have proved to our satisfaction that this method, which we have used exclusively for over ten years, is more anatomically and physiologically correct, speedier, and less disabling by far than any other method with which we have had experience.

FRACTURES OF THE NAVICULAR BONE

Cases Treated by the Method Described	19	Cases Treated by Other Methods*	9
Average period of immobilization....	9 weeks	Average period of immobilization....	3½ weeks
Number of above patients working throughout treatment.....	11	Number of above patients working throughout treatment.....	None
Average loss of time of remainder of above patients....	15½ days	Average loss of time from work.....	12 weeks
Average time from application of cast to discharge.....	10 weeks		

* These cases examined but not treated by us.

SUMMARY

First, a brief description of the anatomy of the carpal navicular bone is presented with a discussion of the probable mechanism of fractures and the points in diagnosis.

Second, a method of reduction and splinting of carpal fractures, which gives excellent results in conjunction with minimum disability, is suggested.

INJURIES TO THE HAND

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THE treatment of injuries to the hand is of vast economic importance. Certain employments have special hazards: in some the eyes are liable to injury, in others the feet, legs, knees, back, lungs, elbows or shoulders, but in a great many the hands are apt to be hurt. By the same token, an individual with a permanently maimed hand is incapacitated for doing so many kinds of work that it may be difficult, or impossible, for him to secure a job. This sad plight may befall the uneducated laborer or the skilled workman. The surgeon who undertakes the care of an injured hand, therefore, assumes a major responsibility. To discharge this responsibility in most cases he need not be a master surgeon. He should have some surgical ability and should possess a good working knowledge of the anatomy of the hand and wrist and a practical understanding of the repair processes of skin, tendon, nerve, and bone. A comprehensive knowledge of the anatomy is not necessary, but it cannot be gainsaid that the more extensive is his knowledge of the subject, combined, of course, with judgment and reasonable operative technique, the better will he discharge his responsibility, the more satisfactory will be his results, and—what is no little thing—the greater will be his joy in the practice of medicine.

It is impossible in a short article to set forth the treatment of all types of injury to the hand. The problems which they present are almost legion. Rather than make a sketchy presentation of many phases of the subject, the writer has chosen a few acute conditions, outlining the general principles of treatment and suggesting some details of technique which have been satisfactory in his experience.

GENERAL CONSIDERATIONS

At the start two old adages come to mind, "tall oaks from little acorns grow" and "an ounce of prevention is worth a pound of cure." These may be paraphrased: Prompt attention to minor cuts may avert serious consequences—and—what is done when a case is first seen frequently determines the course and outcome. Errors of omission or commission at the first treatment of a relatively simple injury to the hand may lead to consequences so serious that the functional result, even after a long period of illness with one or more operations, may be pitiful.

1. *Armamentarium.* "A carpenter is known by his tools." It is impossible to do fine work without appropriate instruments in good condition. The accompanying illustration (Fig. 1) shows a few special instruments which are used to supplement the usual dissecting kit. Most of the work is done by sharp dissection. It causes less trauma than the crushing of scissors. For the same reason no smooth forceps are employed. The magnifying lenses can be clasped over spectacles and are especially serviceable in dissecting out neuromata.

2. *Preoperative Examination.* A careful examination should be made in all cases, unless precluded by extreme pain or shock, to permit an intelligent understanding of the damage before anesthesia is started.[9] With practice and gentleness this can be accomplished quickly and with very little discomfort to the patient. *The repair of a lacerated wound, even an extensive laceration of the volar surface of the wrist, should not be an exploratory operation.*[11] This does not imply looking *into* the wound. It implies covering the wound with sterile gauze and

testing the function of the parts. The surgeon executes with his own hand the movements which he wishes the patient to attempt. Beginning with the thumb, flexion and extension of each phalanx of each digit is tested. To this end it is helpful to support the joint proximal to the phalanx which is being tested. These are gross movements. The muscle bellies of these tendons are innervated in the forearm. To determine the integrity of the nerves at the wrist, the intrinsic muscles, which execute the finer movements of the hand, must be tested. The ability to carry the index, ring and little fingers *toward* the middle finger, or these fingers *away from* the middle finger or the middle finger from side to side; the ability to carry the little finger away from the ring finger, or the ability to carry the thumb to the side of the palm indicates the intactness of the ulnar nerve at the wrist or of its distal motor branches. The ability to appose the thumb to the tips of the fingers or to abduct (not extend) the thumb indicates the intactness of the median nerve at the wrist or of its distal motor branches. Although tests for cutaneous sensation may be of value in estimating the repair problem in lesser injuries and in reconstruction work on the hand, the writer believes that the mental attitude of the patient often renders them unreliable in extensive acute injuries. The rôle of the interossei in flexing the fingers as a whole and of extending the last two phalanges in conjunction with the lumbricales, especially the first lumbrical in case of the index finger, should be borne in mind.

The necessity for thorough preoperative examination is well illustrated in multiple wounds. The wounds which seem trivial may be serious. Apparently insignificant wounds of the wrist may sever the median or ulnar nerves without injury to contiguous tendons. Unless special tests for these nerves have been made, their injury will be overlooked when operation for other wounds is undertaken. Subsequent neurorrhaphy will be necessary. It is needless to state that immediate repair of a nerve is not only much less difficult but also more likely to succeed.

The integrity of circulation may be determined by noting the radial and ulnar pulses at the wrist, the color and warmth of the hand, and the capillary blush in the nail-beds.

3. *Anesthesia.* Certain operations on the digits can be satisfactorily performed with novocaine nerve block. Occasionally evipal is used. In most cases, however, ether or gas-oxygen will be necessary. The author has had no experience with novocaine nerve block at the wrist.

4. *Preoperative Preparation.* The preparation for immediate operation is very important. A hasty or rough preparation may ruin a pretty operation. In cases of severed tendons the part is held during this procedure so as to prevent their further retraction. In the preparation of bad lacerations or crushes a very thorough but gentle cleansing with soap and water is followed by prolonged douching with several quarts of sterile salt solution and completed by a douching with ether. During the process frayed tissues which are floated by the douche are snipped off and damaged tissues debrided. Thirty minutes devoted to such cleansing is profitably spent. "Patience is a virtue." *Impatience to operate is too common a failing.*

Gloves, instruments, and under-sheet are discarded. The *skin* which is to be exposed is painted with half-strength iodine or one of the acetone-alcohol mercurials, and sterile drapes are placed. The hand is elevated several inches on a folded sheet or towels. For reconstruction work the hand and forearm receive twice daily preparations with soap, water, ether and alcohol followed by a sterile dressing, for one or two days before operation. Shaving is done only at the last preparation

The indications for antitetanic serum (or gas bacillus serum) and the technique of preliminary intradermal injection for testing sensitivity are the same as for wounds elsewhere in the body.

5. *Tendon Stitch.* A number of methods of suturing tendons have been devised.[1–6] A tendon stitch which the writer[7,8,11] has used with satisfaction since 1909 is illustrated. (Fig. 2.) It is of No. 7 black silk, once boiled, threaded on a fine, non-cutting, straight or curved needle. It is a simple overcasting affair starting on a tendon border about the width of the tendon, or a little more, from its line of division. The needle is inserted from the underside and a single knot tied. A few overcastings are then made to, but not through, the severed surface. The other margin of the tendon end is similarly supplied with a stitch. The distal portion of the severed tendon is then supplied with marginal stitches. This illustrates an important advantage of the stitch. As tendon ends are identified they can be supplied with stitches and laid aside. When the deepest tendons have been so treated, tying together of the tendons can be done from within out. This method minimizes the operative trauma. Again, the stitch does not penetrate the core of the tendon at its severed surface. Core-penetrating sutures provoke more scar between the tendon ends. The scar may stretch, producing a little slack which defeats complete restoration of function.

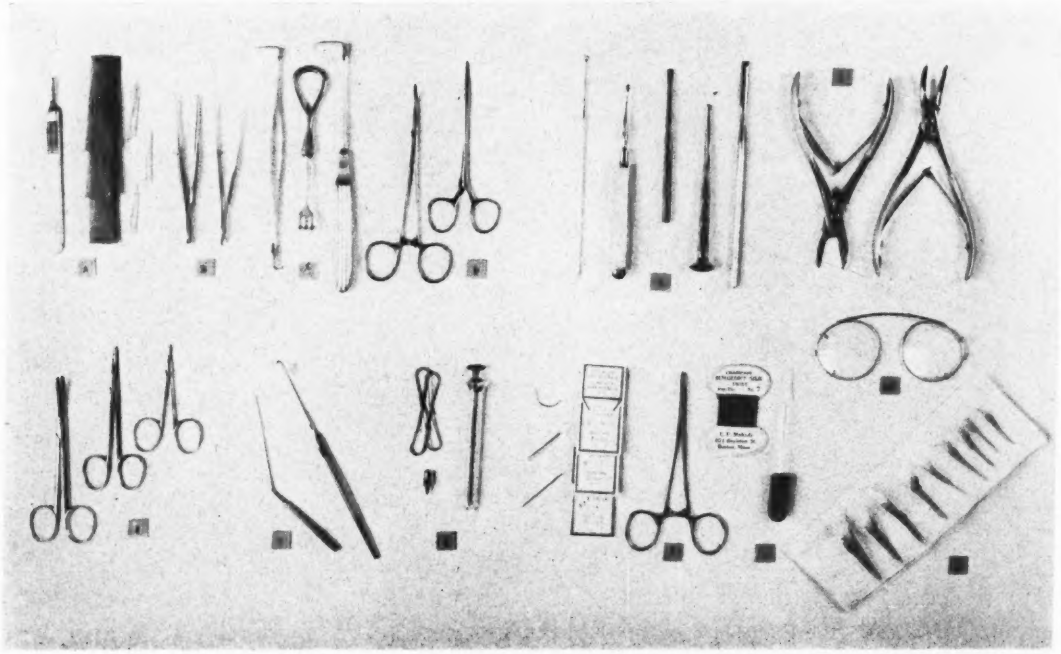


Fig. 1. Armamentarium. Fine instruments used to supplement the usual dissecting bit. A, knife handle and blades. B, fine toothed forceps. C, retractors. D, hemostats. E, scissors. F, fine hooks used in skin grafting for retraction of the margins of skin defects. G, 1 c.c. syringe and rubber bands for digital nerve block. H, needles, holder, and silk. J, dropper for neurorrhaphy. K and L, fine bone instruments. M, magnifying lenses which may be clasped over spectacles. N, vaselined gauze drains.

6. *Tenorrhaphy and Neurorrhaphy.* The bête-noire of tendon surgery is anchoring adhesions. These may be largely prevented by minimizing operative trauma and by early use. Operative trauma may be minimized by preoperative diagnosis, adequate exposure, and gentle and deliberate technique. Attempting to effect a tendon suture through the original traumatic wound almost always means rough handling of the tissues. A generous incision should be made. To reach into a tendon sheath with an instrument to deliver a tendon bruises the tissues. If flexing or extending the finger or wrist does not thrust the tendon-ends into view, the incision should be enlarged. No attempt is made to close tendon sheaths. The additional surgical trauma and suture material creates adhesions. The sheaths heal spontane-

ously. No tourniquet is used. Slight proximal or distal pressure suffices to control bleeding until the incision has been made through skin and fascia. Vessels are then secured. Practically no further bleeding is encountered. The tendon ends are sewn together as above described. The fascia is always loosely closed to cover the tenorrhaphy, even in the fingers. The skin is approximated with fine silk.

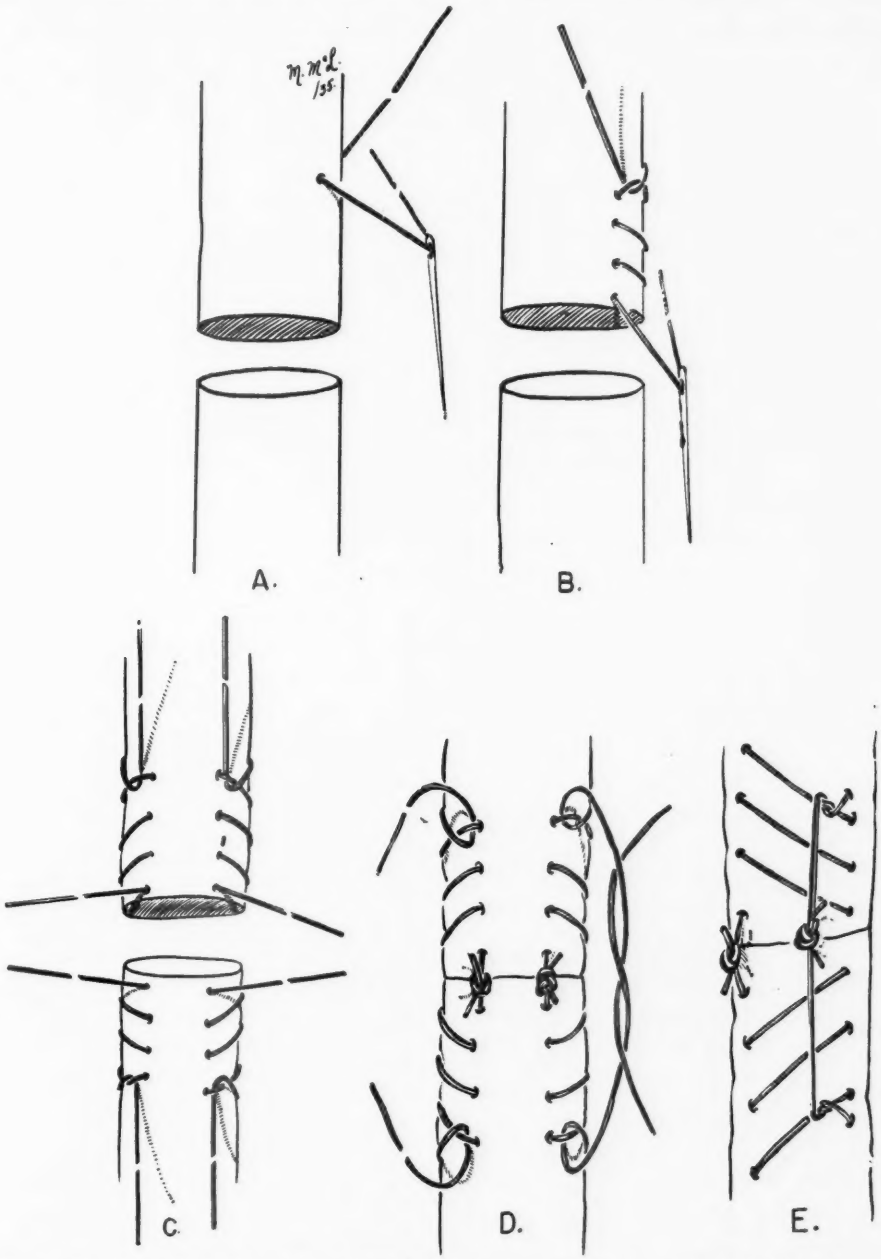


Fig. 2. Tendon stitch. The size of the silk has been exaggerated. A, start of stitch from under surface of tenson. B, single knot and overcasting down to severed surface which is not penetrated. C, both ends of severed tendon provided with stitches ready to tie. D, shorter stitch ends tied and longer ends being tied. E, side view of tendon with stitches tied. The upper surface of the tendon is to the left. (From Harmer, in *New England J. Med.*, 214: 613, 1936.)

Nerves may be differentiated from tendons in fresh cases by their grayer color, the fibered appearance of their sectioned ends, and in the case of the median nerve at

the wrist, by its tiny anterior vessel. If the nerve ends are found frayed, they are cleanly cross sectioned with a small sharp knife. Suture, as described below, is effected with a tiny, smooth, curved needle carrying fine silk. Wrapping the suture line with fat is never practiced by the writer in cases of immediate repair but is done in those cases of later repair in which much scar tissue is encountered. From the time neurorrhaphy is started the part is held in a relaxed position until a splint is applied which maintains this position.

Immediate Repair of Extensive Laceration on the Volar Surface of the Wrist. After the damage to tendons and nerves has been determined by preoperative examination, exposure may be gained by utilizing part of the traumatic wound and in making a distal incision from the root of the palm down to it and a proximal incision from its median or ulnar extremities. At times, the traumatic wound may be transected by an incision from the root of the palm proximally several inches. The median nerve at the wrist is identified just beneath and lateral to the palmaris longus tendon. If the latter is absent, the nerve is very superficial. The annular ligament is carefully divided, for the nerve lies immediately beneath it. The wound margins are retracted. Nothing is disturbed. The structures are studied in situ and identified. It is to be remembered that the tendons of the flexor sublimis digitorum converge to pass through the narrow gutter beneath the annular ligament, those of the middle and ring fingers passing over those of the index and little fingers. It is also to be remembered that the ulnar nerve at the wrist is lying just lateral to the tendon of the flexor carpi ulnaris, that it hugs the pisiform and passes across the annular ligament.

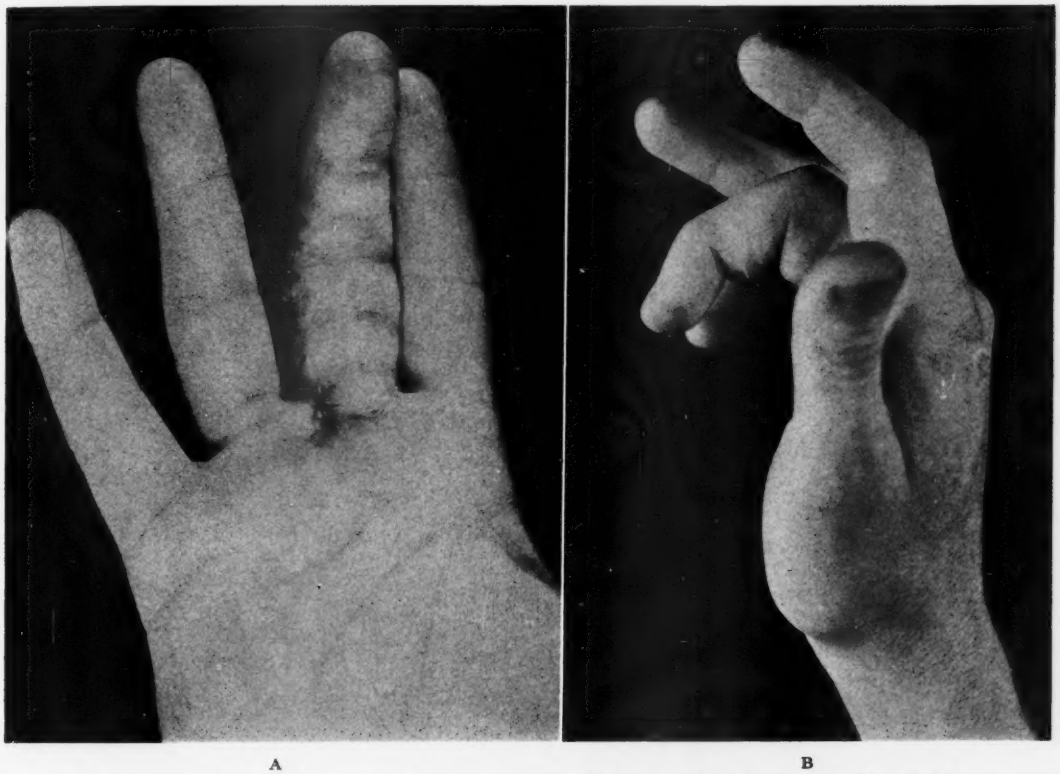
The proximal and distal ends of the median and ulnar nerves are turned out of the wound and wrapped in tiny sponges soaked in warm saline. The ends of the superficial tendons are then provided with stitches, those of each tendon end being clipped together in a small hemostat and laid aside. The deeper tendons (flexor longus pollicis laterally and flexor profundus digitorum medially) are then studied in situ, identified, and similarly provided with stitches. While the wrist is held in a flexed position by an assistant, the tendon ends are tied together from within out, care being taken to secure snug and accurate apposition. The ends of the palmaris longus are not united until the median nerve has been sutured.*

The severed ends of the median and ulnar nerves which have been laid aside in saline soaked sponges are now turned into position. If frayed, they are prepared for suture (as above). The wrist is held in a flexed position. The edge of the sheath of the proximal end of the median nerve, close to its little vessel, is caught with a tiny curved needle, and the silk pulled through. The needle is then passed through an homologus point of the sheath of the distal end. The sectioned surfaces are moistened with salt solution from a medicine dropper and the suture is carefully tied so as to approximate the little vessel. In this way the nerve ends are brought together without twisting the nerve. The stitch ends are clipped in a small hemostat which is used to steady the nerve while the second stitch is placed on the opposite side of the vessel.† Before being tied, the nerve-ends are again moistened with a few drops of saline. These stitches are also clipped in a snap which is used to rotate the nerve so that a stitch may be placed on the side of the nerve and tied under saline drip. In this manner the circumference of the median nerve is approximated by five or six sutures. The ulnar nerve is similarly repaired but twisting must be avoided as

* It may be helpful to the surgeon who is trying this method for the first time to leave the stitch ends sufficiently long so that all those belonging to each tendon may be snapped together and laid aside. After the deepest tendon ends have been tied together, this may obviate the possible necessity of reidentifying the more superficial ones.

† In order to avoid bruising the nerve while the first stitch is being placed, it may be found helpful to steady it gently with a saline-soaked cotton pledget held in a snap.

much as possible without the guide of a tiny vessel. Flexion of the wrist is maintained while the tendon ends of the palmaris longus are tied together, the annular ligament and fascia are loosely approximated with fine catgut, and the skin is closed with silk, and until a splint is applied.



A B

Fig. 3. Tenorrhaphy done three weeks after severance of flexor tendons by heavy glass. Sublimus tendon excised, profundus repaired. Picture eleven days later to illustrate active motion and skin incision. Stitches were retained until photograph was taken. For clearness, the proximal phalanx is not being supported by patient's other hand during exercise. (See text.)

No drainage is used. If a wound is so soiled that drainage is necessary immediate tenorrhaphy and neurorrhaphy should not be attempted. This is a safe rule. Such a wound should be cleansed and Dakin's treatment instituted. The sheaths of the nerve ends may be caught together with a stitch. This facilitates subsequent repair. Tenorrhaphy and neurorrhaphy should not be practiced for some weeks after complete wound healing has occurred. The splint, however, may be worn for a couple of weeks to minimize the retraction of tendons.

7. *After-Care.* The objective of all repair procedures on the hand is the restoration of function. In tendon surgery, as previously stated, the prevention of adhesions is of vital importance. This may be accomplished in part by minimization of operative trauma and in part by the institution of early function. Prolonged splinting following tenorrhaphy is to be avoided. Within a few days after operation systematic, active (not passive) motions are started. Each phalanx of each digit is slowly and deliberately moved several times every few hours. Use of the sutured tendons is encouraged up to the point of pain, but forcible use of the antagonistic muscles is discouraged. (Fig. 3.) When exercising a phalanx the patient steadies

the phalanx proximal to it. To curb the over-zealous individual, rest between exercise periods is exhorted.

finger). At night for a couple of weeks, however, in case of the repair of flexor tendons, the fingers are held flexed into the

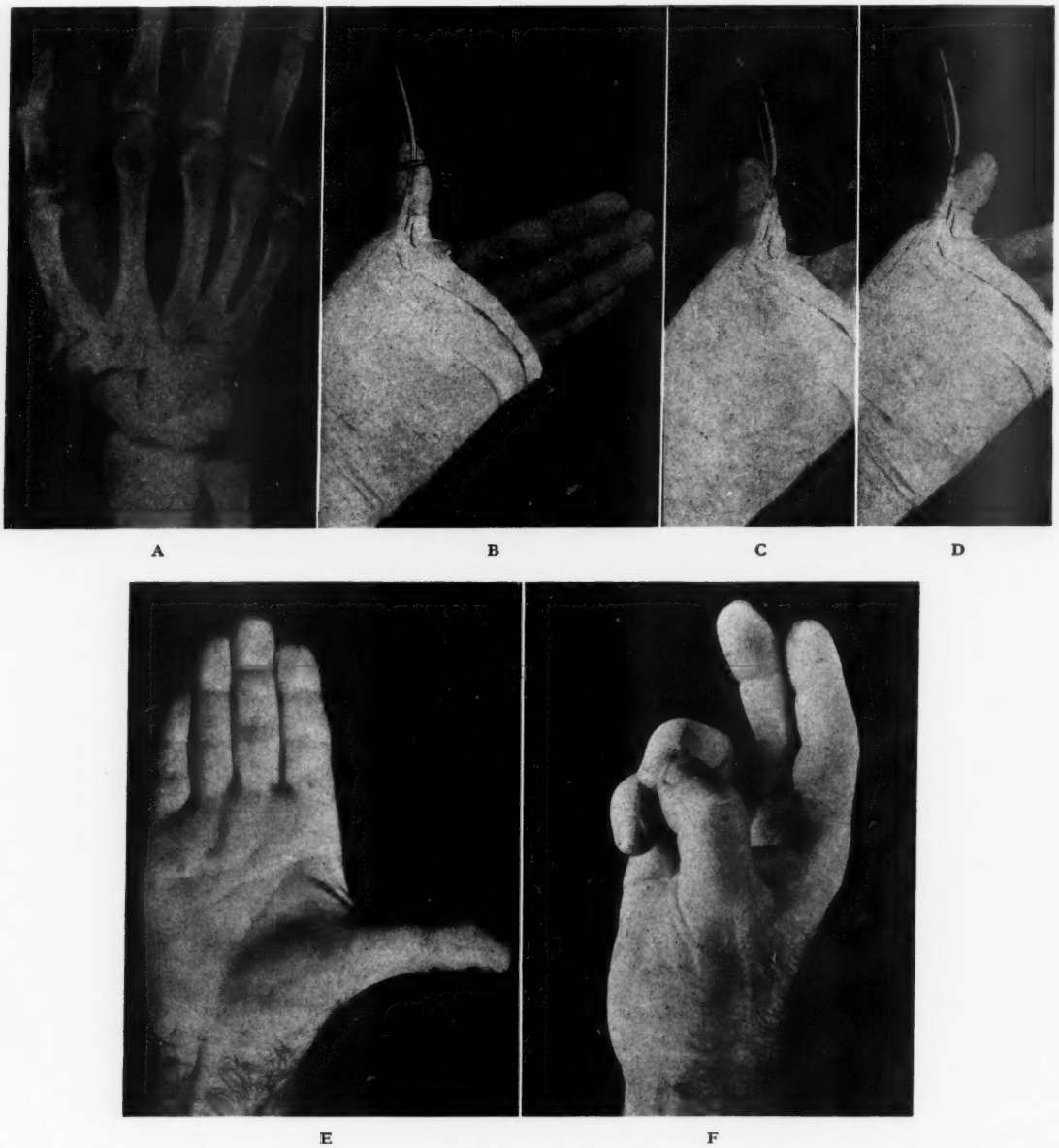
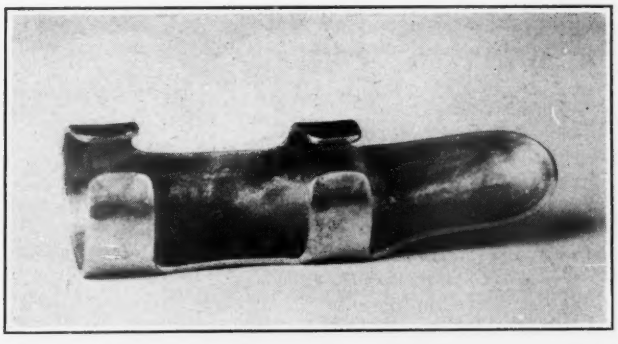


FIG. 4. A, compound comminuted fracture of the first metacarpal bone, severance of extensor tendons of thumb and laceration of abductor pollicis muscle. Immediate repair after prolonged preparation (see text). B, banjo type of splint used to resist antagonistic pull of long flexor with rubber band. C and D, rubber band pushed up to permit active exercise. Pictures taken thirty-seven days after operation. Plaster (bulkier than usual on account of fracture) and skin stitches removed same day and figure-of-eight adhesive strapping to metacarpophalangeal joint applied. E and F, seventeen months after operation to illustrate function of thumb. Note traumatic and operative scars.

Following tenorrhaphy without coincident nerve repair no splinting other than that afforded by the bulk of the dressing is used, except in case of repair of extensor tendons in the terminal phalanges (see drop

palm by a towel laid over the hand and loosely secured about the wrist and, in case of the repair of extensor tendons, a volar splint is secured to the hand and forearm by buckle straps or a bandage. Following

tenorrhaphy of the extensor tendons of the thumb or of the fingers at the knuckles special devices are employed to restrict the pull of antagonistic muscles in both classes of cases is practiced for three weeks.[11] At night, for another two weeks, the



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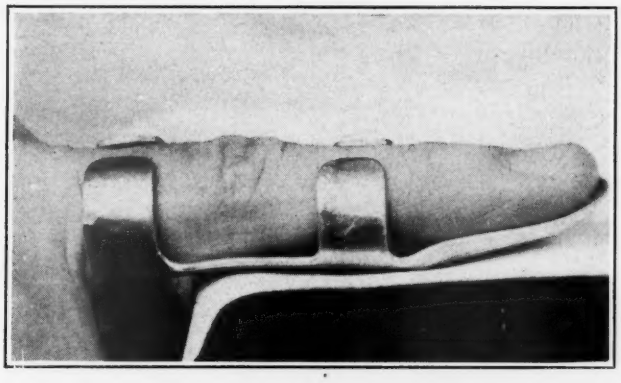


B

Fig. 5. Digital splint of aluminum used to hold terminal phalanx in extension (to eliminate the antagonistic pull of flexors) following repair of extensors of thumb and avulsions of extensors in terminal phalanges. (From Harmer, in *New England J. Med.*, 214: 613, 1936.)

pull of antagonistic muscles.[10] If the extensor tendons of the thumb have been sutured, a small banjo splint with rubber band support for the terminal phalanx is used.[11] (Fig. 4.) This device prevents the pull of the strong flexor tendon, but by removal of the rubber band permits active use of the long extensor. After the wound has healed (if the metacarpal is not coincidentally fractured) the metacarpophalangeal joint is stabilized with a figure-of-eight adhesive strapping and the terminal phalanx supported by a metal gutter splint (Fig. 5) which can be easily slipped off for exercising the sutured tendon. A similar banjo device is employed in the treatment of extensor tendons which have been sutured at the knuckles. Resisting the thumb cases wear the small metal splint and the knuckle cases a simple volar splint which maintains the fingers slightly hyperextended.

Following neurorrhaphy, with or without coincident tendon repair, splints are always employed; first, to prevent strain on the nerve until regeneration has advanced sufficiently to endure such tension, and later, to prevent the pull of antagonistic muscles on the atrophying muscles. For example, following neurorrhaphy with coincident tenorrhaphy at the wrist, a simple splint (Fig. 6) is employed.[8] This device permits the active use of the coincidently sutured flexor tendons but by maintaining the wrist in flexion prevents pull on the sutured nerves and by holding the fifth and

fourth fingers in flexion at the metacarpophalangeal joints prevents the stretching of the atrophying hypothenar muscles (in case of ulnar neurorrhaphy). After reading Mason's paper[12] in which he pictures his "wrist strap and thumb loop" to relax the thenar muscles, the author added a thumb flange to serve this purpose. More recently he has been using a cotton suede thumb cot (Fig. 7) in conjunction with the original metal splint. (Fig. 6.) This device is worn for two months. If it is taken off occasionally by the surgeon and the wrist gently moved, no stiffness will occur. After this time, it is presumed that nerve regeneration has sufficiently progressed to withstand tension at the wrist. The desideratum then is to prevent the stretching of the atrophied muscles by restricting the pull of antagonistic muscles. In case of the median nerve, Mason's "wrist band and thumb loop" or the author's "thumb cot" may be used. If the ulnar and median nerves, or the ulnar alone, have been sutured at the wrist, the "flanged cross splint" (Fig. 8) is recommended.

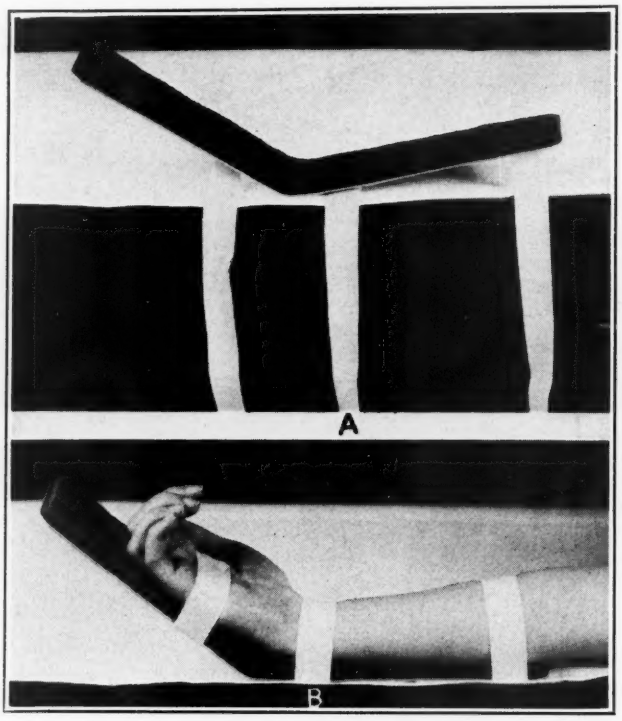


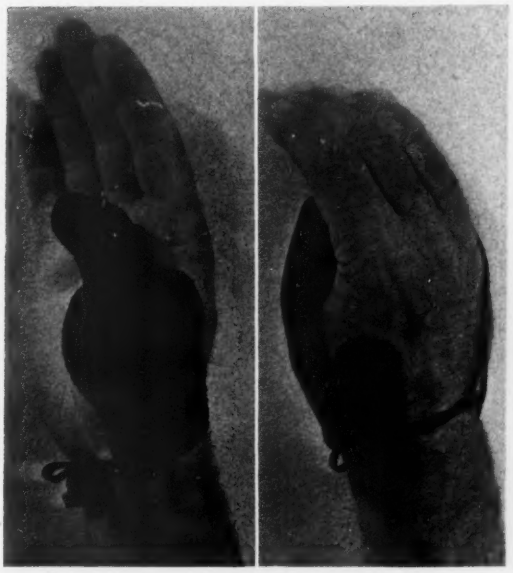
Fig. 6. Wrist splint of felt-covered aluminum used following repair of nerves and flexor tendons at wrist. A, the splint has been gently angulated to receive the flexed wrist. B, splint in use. Although the fingers can be actively moved, tension on the suture nerves is prevented. The support of the fifth and fourth metacarpophalangeal joints prevents undue stretching of hypothenar muscles in case of ulnar neurorrhaphy. The addition of the thumb cot (Fig. 7) similarly protects the thenar muscles in case of ulnar or median neurorrhaphy. The splint does not fit the subject, as the forearm portion is too short and the hand portion too long. (From Harmer, in *New England J. Med.*, 214: 613, 1936.)

8. *Late Tenorrhaphy and Neurorrhaphy.* Wide delicate dissection through scar tissue is necessary for the identification and mobilization of structures. All scar between tendon and nerve ends must be removed and the ends themselves prepared for suture by clean transection with a sharp knife. (Fig. 9.) The proximal end of a nerve

is usually sought first because it can be recognized by its "bulb." The distal end possesses no label. Tiny sections of the nerve ends should be removed until both present normal appearing surfaces. This must be done conscientiously. Fear of creating a sizeable gap between nerve ends is, the author believes, the commonest cause of failure. *Nerve regeneration is impossible through scarred ends regardless of the perfection of their suturing.* The author also believes that end-to-end approximation without undue tension must be effected and has little confidence in the use of tubes (sections of veins, etc.) to bridge defects. A section of sural nerve, as suggested by Bunnell[3] may be used to bridge the defect, serving as a conduit for the advancing nerve fibers.* At the wrist flexing the hand and elbow may suffice. If not, the incision must be extended proximally and the nerve carefully freed for a sufficient distance. If approximation cannot then be effected in case of the ulnar nerve, the nerve may be transplanted anteriorly at the elbow. This has worked beautifully in three cases.

If gaps between tendon ends are too great to permit approximation, tendon grafting or transplantation may be practiced. For grafting the author prefers excised tendon (palmaris longus, plantaris, strip of achilles) to excised fascia. The graft must be somewhat shorter than the defect. The grafting of extensor tendons is simpler than that of flexor tendons. If tendon transplantation is feasible it is technically less difficult than grafting. An overlying skin defect must be closed with a skin-and-fat graft (pedicle, rope, tunnel) before tendon surgery is undertaken.

9. *Finger Amputation. Painful finger stumps are common and are often due to hasty operative technique.* Secondary operations for neuromata or reamputation should seldom be necessary. Although finger amputation is a simple procedure, it should be done with precision. The desideratum is a well-padded, non-sensitive stump.



A B

Fig. 7. Thumb cot of cotton suede to relax the thenar muscles. Used early in conjunction with aluminum wrist splint (Fig. 6) for neurorrhaphy and tenorrhaphy at wrist and later in those cases in which only the median nerve has been sutured (see text). Note double lacing from tip carried between fourth and fifth fingers to back of wrist, looped, carried through hem at base and tied about wrist.

If the nature of the injury permits, a long anterior and a short posterior flap should be made to bring the resulting scar on the dorsal aspect of the stump. A simple method is as follows: With a rubber band tourniquet and under novocaine block, a mid-lateral incision is made on each side of the finger. The distal ends of these incisions are connected by a straight transverse incision through the volar soft parts and the proximal ends by a transverse incision through the dorsal soft parts. The flaps are separated from the bone on all sides, turned back and held with a wrap of gauze. This is preferable to the trauma of tenacula. The bone is then bitten through at the desired level. The bone end is carefully rounded off with small rongeurs and curette. (Fig. 1.) The digital nerves are sought, seized with tiny hemostats, separated from adjacent

* The author has not had occasion to resort to this procedure at the wrist, but has used it with promising results in a relatively recent case to bridge a 3½ inch defect in the peroneal nerve.

tissues as far proximally as possible and cut off. The flaps are then folded over the bone end and, if too long, are shortened by straight transverse incisions. The flaps should, however, always be of generous length, especially the volar one. The elastic tourniquet is cut and bleeders secured with 00 plain catgut. The subcutaneous portions of the flaps may be caught together with not more than two or three sutures of the same material. The flexor and extensor tendons are not sutured over the bone end. The skin is closed interruptedly with not more than five or six fine silk stitches. Care is taken to secure perfect approximation at the sites of suture. If the flaps are sufficiently generous to permit natural shrinking without tension, the writer has never seen a poor cosmetic result from these right-angled corners, "dog-ears." They have always drawn in and given a smooth, rounded, and well-padded stump. Although suturing the tendons over the bone end may be considered a neat means of "padding," the procedure is unnecessary with a generous volar flap, and is, moreover, undesirable. The contraction of the tendons may lead to a pulling sensation either in opening or closing the hand, which may be very irksome in certain occupations.

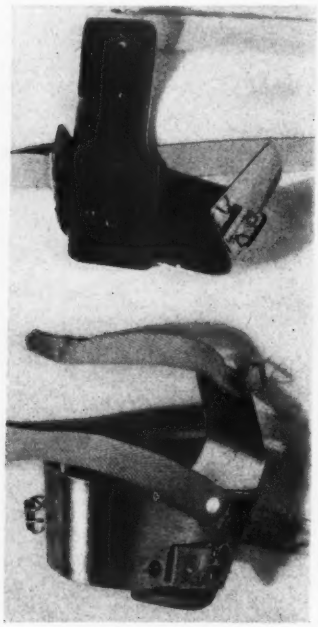
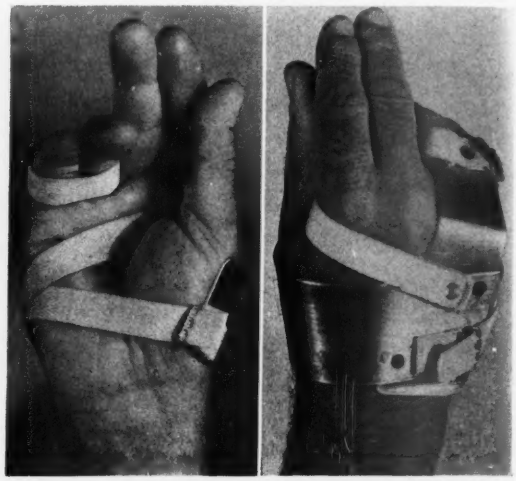


FIG. 8A. Flanged cross hand splint of aluminum used in later treatment of ulnar neurorrhaphy or ulnar and median neurorrhaphy at the wrist or base of palm.



B C

FIG. 8B AND C. The flanged cross hand splint in use. The metacarpophalangeal flange prevents undue stretching of the hypothenar muscles (ulnar). The thumb flange similarly protects the thenar muscles (median and ulnar). The medial flange steadies the hand in the splint. It is lined with felt or applied over a glove from which middle and distal phalanges have been cut. It is readily removable for washing.

10. *Drainage Material.* The author uses only gauze, well smeared with vaseline or boracic ointment. These drains are more or less slender cones made of one or several thicknesses and are prepared at the operation. (Fig. 1.)

INCISED AND LACERATED WOUNDS

The character and severity of these wounds varies from trivial superficial cuts to deep slashes, from slight tears to gougings which may rip open the hand and wrist. In very many cases vessels, tendons, and nerves are severed; in some cases joints are opened and infrequently bones are damaged. If wounds are so soiled that drainage is necessary, immediate repair should not be attempted. (See 6 above.) In the vast majority of these wounds, however, immediate repair without drainage can be practiced. Most wounds should be

FIG. 9. Late median and ulnar neurorrhaphy and tenorrhaphy at wrist. An immediate repair had been attempted elsewhere eight months previously. A and B, ability to flex and extend fingers and thumb, September 19, 1928. Note atrophy of thenar and hypothenar eminences. Operation, September 21, 1928, showed both ends of median nerve sutured to tendons, ulnar nerve unsutured and other tendon ends matted and bridged by scar. C, D and E, ability to flex and extend fingers and thumb in May 1929. F, G and H, September 10, 1938, showing flexion and extension and ability to appose and adduct thumb. Note development of eminences.

enlarged in order to effect tenorrhaphy or neurorrhaphy without undue operative trauma.

In the digits, although the traumatic wound is usually transverse, surgical approach is gained through a mid-lateral incision. When the resultant right-angled flaps are retracted, ample exposure is secured. In the author's experience, if these flaps are carefully approximated, severed digital nerves heal spontaneously.

In the palm these nerves are sought and, if possible, each is approximated with a tiny silk stitch. The motor branch of the median nerve and the deep branch of the ulnar nerve to the thenar muscles, of course, must be sutured. Preoperative examination will have demonstrated their injury. The repair of wounds at the base of the palm requires detailed anatomic knowledge and surgical deftness. Division of the annular ligament may be necessary. In the palm and wrist, in contradistinction to the fingers, surgical incisions for better exposure than that afforded by the traumatic wound should follow as far as possible the natural creases. Certain traumatic wounds, therefore, may be enlarged by lengthening, but others require right angle or oblique incisions from one or both extremities. Some, however, may need transection, but acute angled flaps are to be avoided.

Deep slashes across the volar surface of the *wrist* which divide both the radial and ulnar arteries, the median and ulnar nerves, and most, or all, of the tendons practically always spare the anterior interosseous artery. Through this little vessel adequate collateral circulation must practically always be established.* Tenorrhaphy and neurorrhaphy in this situation are not difficult.

DROP FINGER

This relatively slight deformity may result from a cut or a crush, but is usually due to a closed tear of an extensor tendon at or near its insertion. A small piece of bone may be avulsed. Splinting of the finger with the distal phalanx in hyperextension may, if started within a few days of the accident, give a perfect result (restoration of complete extension without limitation of flexion). Not infrequently more or less loss of extension persists.

Many individuals may be able to "carry on"; others may have their speed of production reduced and their liability to injury increased; a few may find it impossible to continue a highly technical job. To these few, amputation of the terminal phalanx is only the substitution of a lesser evil. They should be offered surgical repair. The author says this in full realization that operative results will be disappointing unless the surgeon is willing to give time and attention to details of after-care.

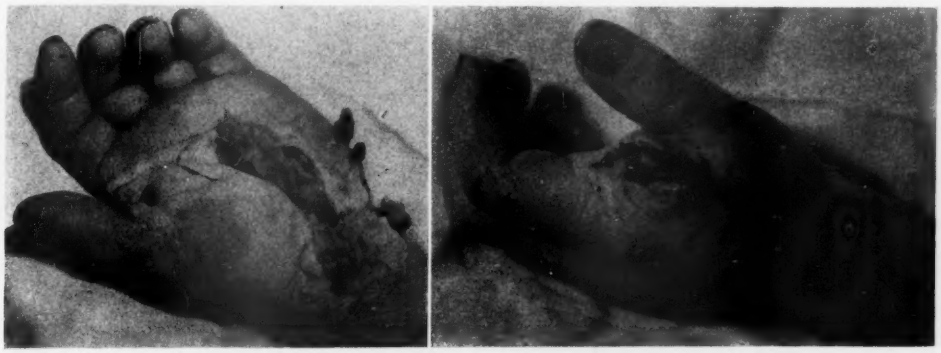
Under novocaine block anesthesia an inverted L incision is made, the short arm crossing just distal to the distal joint, the long arm passing down the middle phalanx in a lateral position. The right-angled flap is reflected with as much subcutaneous tissue as possible. The bone fragment, if present, is removed. No attempt is made to close the joint. The terminal phalanx is held by an assistant in hyperextension throughout the remainder of the operation until a padded volar splint which maintains this position is applied. The tendon is united with a couple of mattress stitches of fine silk. Much suture material is to be avoided. The skin flap is closed with interrupted silk stitches. In late repair in which the tendon-ends cannot be approximated, bridging the gap with silk strands is effective [9] (but the writer now immobilizes a week longer). After the skin stitches have been removed, a metal gutter splint (Fig. 5) is substituted for the wooden splint.

The patient is seen twice a week. The surgeon gently moves the joint to prevent stiffness. The patient is permitted guardedly to extend the phalanx actively once or twice but is forbidden to flex the phalanx. Immobilization is continued for

* The author has never attempted arterial suture in this situation but in quite a number of cases of this character in which tendon and nerve repair has been practiced, he has never seen gangrene of the hand ensue.

three weeks. If the phalanx shows any tendency to drop after forty-eight hours, the splint is reapplied for a week. It is then worn at night for a fortnight.

practically useless. The deeper punctures should at once be judiciously incised. Although this may occasion a few days' loss of work, it will avert many calamities.



A B

Fig. 10. Midpalmar and thenar abscesses with superficial infection over hypothenar eminence and at wrist. A septic hematoma at base of palm had been opened by a physician one week previously. A and B, taken four days after operation (April 6, 1933). Series of vaseline gauze drains in incisions. The annular ligament has been divided to relieve tension on median nerve and tendons beneath it, but drains are placed only to the structures (not between them). No deep wrist infection.

Fig. 10c. Shows condition on September 8, 1933, when patient resumed full duty as a carpenter with no inconvenience. The scar proximal to the wrist was from drainage of a subcutaneous abscess opened April 17, 1933.

PUNCTURED WOUNDS

Tiny wounds from metal chips, fine wire, steel wool, tacks, needles, etc., may occasionally lead to serious consequences. Squeezing to make them bleed is intelligent but, if too zealous, may be harmful by bruising tissue. Painting with iodine is

Incision after a lymphangitis has extended up the arm, especially in the absence of a definitely localized induration, is comparable to enlarging one end of a burrow after the animal has escaped through the other end.

At that stage, massive fomentations should be applied and changed every two

hours until the lymphangitis has disappeared. If the lymphangitis does not subside and if the pulse and temperature

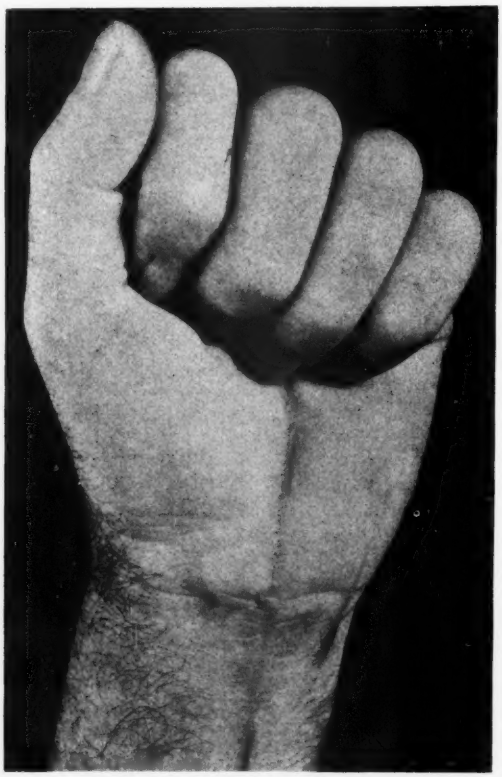


FIG. 10D. Same case on September 8, 1933 with fist closed.

rise, white counts are done, the blood typed for transfusion and perhaps a blood culture is taken. Such patients may suddenly become desperately ill. All preparations should be made for such a contingency.

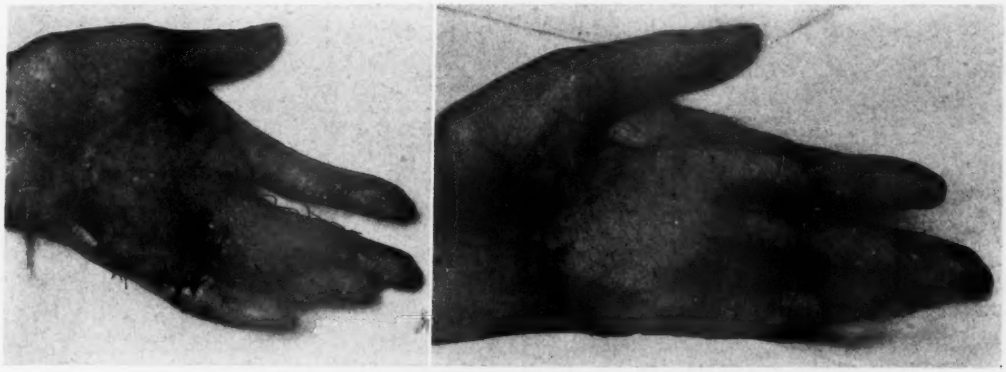
If a definite induration at the site of puncture has developed, it is carefully opened, a culture is taken with a tiny swab and sent immediately to the laboratory. If possible, several individuals who have recently recovered from a hemolytic streptococcus infection should be notified that they may be needed as donors for transfusion. If the laboratory reports a hemolytic streptococcus, a compatible donor is secured and, if the patient is no better, a whole blood transfusion is performed. Theoretically, in immune transfusion the strain of hemolytic streptococcus from which the donor has recovered and that with which the patient is infected should be identical. Practically, in my experience, absolute specificity is not always necessary. If the bloods are compatible, even if the strain of hemolytic streptococcus is undetermined in either individual, it is intelligent practice to use the recently recovered individual as donor for a whole blood transfusion to the patient without delaying the transfusion until it is determined that the donor has been and the recipient is infected with the same strain of hemolytic streptococcus. Hemologic studies may, however, be started which will later prove profitable if transfusion fails. From my experience with immune transfusion, the benefit derived from the donor's blood may not be solely attributable to the specificity of the strain of the hemolytic streptococcus. It would seem that the whole blood of the donor may at times contribute actively phagocytic leucocytes and an opsonin which does not demand absolute specificity of strain. There may be other factors which contribute to the efficacy of such nonspecific transfusions.*[11] In the absence of donors for immune transfusion the bold but carefully checked use of sulfanilamide is advocated. (See also under Infections.)

INFECTIONS

At the start, two statements may be made: an incision for the establishment of drainage of a septic finger or hand should not be a quick slash—and—the administration of sulfanilamide, even in proved hemolytic streptococcus infections, does

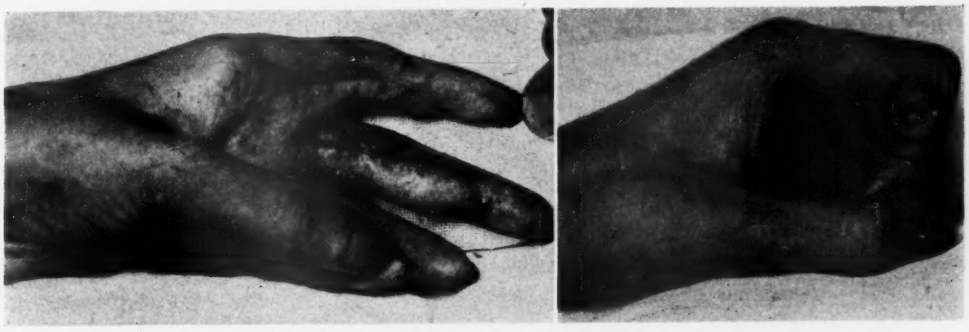
* My first immune transfusion was done in 1930. On April 14 I had operated upon a doctor desperately ill with a hemolytic streptococcus infection of his left index finger sustained by a needle prick. On June 11, I performed an appendectomy with drainage on a child. The culture proved to be a hemolytic streptococcus. Some days later the child went into collapse. I transfused the child with the doctor's blood. The immediate benefit was not less than spectacular. The strain of hemolytic streptococcus was unknown in both donor and recipient. I fully realize that this procedure was not scientific, but the happy result in this case has been happily repeated in others since then.

not supplant surgical drainage.* In infections of tendon sheaths, bursae or fascial spaces, drainage incisions should be made carefully and deliberately. A tourniquet is always used. If a finger sheath is involved, a mid-lateral (never a midline) incision is made. The author has had no experience with the flap incisions of Auchincloss†[13,14]



A B

Fig. 11. Mangling accident five and one-half months before. Entire dorsum of hand except over fifth metacarpal covered by thin, discolored scar. Contracted scar of palm, fingers and base of thumb, which held the fingers in flexion and the thumb into the palm. Diligent massage and exercise were carried on for six weeks before operation was undertaken. A, thirty-three days after grafting of pedicle graft from abdomen, twenty days after division of pedicle, and seven days after division and fashioning of two interdigital bridges. Note ability to extend fingers and adduct thumb. B, taken ten days later, nine days after division of remaining interdigital bridge.



C D

Fig. 11C and D. Taken at same time as Figure 11B. Note mobility of thumb and fingers. It will be necessary later to excise excess fat from middle phalanx of middle finger.

The wound is retracted, the sheath is well exposed and nicked under vision. A culture is always taken. The wound is enlarged in either direction until gentle pressure proximally and distally no longer extrudes pus. If the infection has extended into the bottom of the sheath of the index, middle, or ring fingers, the incision is continued to its bottom, but a bridge of tissue is always left distal to the crease at the base of the finger. The tendons are never dislodged. A series of small greased drains is laid into the wound *to* the tendons and cut off close to the skin with a sharp pair of scissors. The drains are never forced into the sheath for fear of damaging the mesotenons. The tourniquet is removed

* The author has operated upon cases of tenosynovitis after prolonged trials of sulfanilamide had failed. The mesotenons and parietal and visceral sheaths were injected and covered with a slimy exudate. Cultures have been negative but the delicate tissues showed the effects of long continued irritation.

† Auchincloss states: "In the fingers as soon as the skin has been divided, retraction should be done with care and the dissection carried towards the midline. If one incises the sheath from its side dorsal to the digital vessels, it is impossible to avoid cutting the branches running to it. The incision in the sheath should be done in front of the blood vessels and nerves lying on either side. The nerves run slightly anterior to and inside the blood vessels."[13]

and bleeders secured. A sponge, generously smeared with vaseline or boracic ointment, is laid over the wound and the dressing completed.

For several days dressings are done daily. As the innermost sponge is raised with one forceps, another is used to reach under and hold the drains in place. An effort is made not to remove them until they are extruded by the process of tissue repair. The part is cleansed with ether and another well greased sponge applied. Tendon sloughing may result from damage to the circulation in the mesotenons caused by the infection before drainage is established, but it may also be caused by rough surgery in introducing drains. Incisions may occasionally be required on both lateral aspects of a finger. If so, small bridges of annular ligament are left in each phalanx and two series of tiny drains are used. Through and through drains are viciously traumatizing. For drainage of deep infections at the root of the palm, the author never hesitates to divide the annular ligament. This relieves tension on the tendons and the median nerve which are confined in the gutter beneath it. Drains, however, are never forced down between the structures. They are used merely to hold open the wound. Subsequent repair of the ligament has never been necessary. (Fig. 10.) The reader is referred to the classic work of the late Dr. Kanavel "Infections of the Hand"[15] and to contributions by Auchincloss,[13] Cleveland,[16] and Grinnell.[14]

BURNS

Some of the causes of burns of the hand in industry are: contact with hot objects, steam, chemicals, hot oil, fire, acetelyne torches, molten metal, and electrically charged wires. All degrees of burns may, therefore, result: erythema, vesiculation, destruction of skin into subcutaneous tissue, into muscle, or even to bone. Small or large areas of one or both hands may be involved.

Shock, if present, should be appropriately treated. Contact with high voltage wires may demand immediate and perhaps prolonged artificial respiration.

First degree burns require only protection from air with simple (non-carbolated) salves, but most other burns are best treated by surgical cleansing followed by particular methods. In plants which are especially equipped, spraying with paraffin preparations is advantageous. Air is excluded and, if the spraying is done immediately, the burn itself may be considered sufficient sterilization of the wound. The wax possesses no antiseptic or coagulating properties. Early compensation for fluid loss by saline and glucose, especially in the presence of coincident burns elsewhere, should not be neglected. If this method of treatment is continued it keeps the granulations in good condition for grafting.

Other methods demand initial surgical cleansing. This is greatly facilitated if grease is not used as an emergency dressing. Nurses in plants, instead of using carron oil, can just as well cover the part loosely and generously with sterile sponges soaked in warm saline, wrap the hand and arm in sterile towels, place on a wooden splint (without bandage or adhesive straps) and do the whole up in a light weight blanket. This is a satisfactory transportation dressing. For the occasional chemical burn, the hand may be immediately submerged in warm running water into which *later* is dumped a large quantity of alkali or acid (partially dissolved soda bicarbonate or vinegar). Industries which use chemicals will have on hand appropriate neutralizing agents.

Primary surgical cleansing may be done under a sedative or an anaesthetic and should be gentle but thorough. Soap and water, followed by saline and finally by ether, are satisfactory.

The coagulating (tanning) methods of Davidson[17] with tannic acid, or of Bettman[18–21] with tannic acid followed by silver nitrate, may then be employed. Thorough drying under aseptic precautions is imperative (electrically heated cradle and sterile sheets). The coagulum will

peel as epithelialization takes place. Third and fourth degree areas will not heal, however, under a coagulum unless some islands of skin remain. These areas from deeper burns may soften beneath the eschar or the eschar may become so adherent that it will need to be loosened under saline packs or by cutting. In cases where the fingers are burned, the author prefers to use gentian violet because of the more pliable eschar. It will adhere, of course, to more deeply burned areas, but can be eased off after several applications of saline soaked sponges. By this time very gentle active motions can be started.

Those areas which have not epithelialized may be variously treated. Ointments are in order (strips of wide meshed gauze impregnated with boracic acid ointment with or without cod liver oil, Bettman's ointment[19,20] etc.). If spontaneous closure will not lead to contractures, nothing further need be done. If skin grafting must be practiced, Reverdin grafts require less technical skill, but for the hand are not serviceable and are unsightly. If all of the deep scar be removed and the skin edges of the defect gently mobilized, a nicely executed Ollier-Thiersch graft is satisfactory, even over finger joints. Patience and nice technique are as important in the after-care as in the operation. For an extensive area in the palm a pedicle graft from a non-hairy portion of the body may be preferred. Here again, nice technique is imperative (complete excision, neat fitting of the flap, avoidance of excess subcutaneous fat). The author has had no experience with the use of ferric chloride nor with dyes other than gentian violet. His experience with high voltage burns has been limited to reconstruction surgery on hands so maimed.

For those cases in which contractures have developed much can be accomplished by judicious massage and exercise. For lesser contractures of the fingers a Z transposition of flaps may be feasible. If not, a tunnel graft on the medial aspect of the other arm is very satisfactory for one or two fingers. In cases in which the palm or dorsum, as well as the fingers, is involved, a pedicle graft or a rope graft with subsequent division and fashioning of the interdigital bridges may be practiced.

FRACTURES

Fractures form another important phase of this subject which cannot be more than mentioned in passing. Striving for a perfect anatomic position is a laudable aspiration, but restoration of function should always be the cardinal objective. A hand with perfectly aligned fractures but with partially ankylosed joints is a poorer economic result than one with less perfect reposition but with good working joints. In other words, in striving for alignment in fractured phalanges or metacarpals by the use of skelatal traction with small pins, for example, enthusiasm over post-reduction *x*-rays should not postpone the institution of early motion. In fact, many cases of finger and metacarpal fractures can be treated satisfactorily in a flexed position over a roller bandage. Some may be benefited by a few days of preliminary traction.

Fractures of the scaphoid, however, require immobilization for eight weeks in a plaster cylinder. If this becomes loose, it may be replaced by a snugly fitting leather brace with metal reinforcement anteriorly and lacing posteriorly. A dowel graft after the method of Murray[22] usually overcomes pain and weakness from ununited fracture of this bone. Painful wrists following comminuted fractures of the carpus (severe falls or crushes) may be satisfactorily treated by fusion in the position of optimum grasp with shortening of the extensor tendons.

TRAUMATIC AMPUTATIONS

Under this heading are assembled cases with loss of tissues by (1) more or less transverse sections (heavy blades, saws, etc.); (2) tangential sections (planing and buffing accidents); and (3) circum-

ferential wounds (wires, chains, rings, etc.). They may involve one or more digits, the metacarpus, or the carpus. The surgical problem is determined by the magnitude and location of the injury, the damage to circulation and the extent of skin loss. The thumb should never be primarily sacrificed unless it is absolutely hopelessly damaged. Conservative surgery should also be practiced on the fingers, especially if several are injured. The entire proximal phalanx, or as much of it as possible, should be preserved. The loss of this phalanx seriously weakens the power of grasp.

The simplest cases in the first group (more or less transverse sections) are those in which just the soft tip of the finger is cut off. The piece may rarely be reapplied as an autogenous graft or, after thorough cleansing, the defect may be allowed to close spontaneously within a protective cylinder. In most instances a finger is completely transected through a bone or a joint. If an attempt is made to close this surface, or if it is permitted to close spontaneously, a painful stump always results. A modified amputation should be done, sacrificing as little skin as possible. A short, mid-lateral incision may be made on each side extending proximally from the sectioned surface, and the procedure completed as previously suggested (see section 9 above) removing as little bone as will give a well-padded stump but carefully cutting back the digital nerves. In some cases several fingers may require such treatment. At times a phalangeal bone may be divided, while the distal and proximal portions of the finger may be attached by an isthmus of soft parts including the vessels. The bone may be very slightly shortened and this isthmus with some of the soft parts of the distal portion may be used as a flap to cover the end. If, however, such a freak accident occurs to the index finger in association with the complete severance of a couple other fingers, it may be good judgment to try to save the freak finger by *very* thorough cleansing, immediate or delayed tenorrhaphy, loose skin closure and splinting.

In the second group (tangential sections) the skin and tendons may be lost and the bone shaved down. One or more phalanges of a finger may be involved, but usually several. Either the volar aspect (planing accident) or the dorsal aspect (buffing accident) may be injured. If one or two fingers are concerned, especially if they be the little and ring fingers, immediate amputation of the damaged portions may be the best treatment. If several fingers are involved, including the index and middle fingers, it may be deemed justifiable to attempt to save them, but to amputate the lesser fingers. The raw areas are to be cleansed and dakinized until the granulations are suitable for skin grafting. The graft must provide a skin and fat coverage to permit subsequent tendon grafting. If the volar aspect is involved, a tunnel graft from the inner aspect of the other arm is convenient. If the dorsal aspect is involved, the abdomen or thigh may serve as donor sites. Throughout the period until the tendon grafts are functioning, the finger joints should be kept supple and stiffening from the pull of antagonistic tendons combated. If the palm or dorsum is involved as well as the fingers, a pedicle or rope type of graft may be employed. The fingers must be held apart so that the interdigital bridges, when divided and fashioned, will give easy coverage for the fingers.

In the third group (circumferential wounds) are included cuts and tears caused by loops of wire, cords, chains, or by rings which more or less surround the part, usually a digit, but at times the metacarpus or carpus. Fractures may or may not be present. A digit may be partially or wholly avulsed. The whole hand may be torn off. In all cases thorough cleansing and débridement without closure are indicated. In many of the incomplete digital injuries the entire loss of circulation demands prompt amputation with secondary closure. But, if there is even a meager

possibility of circulatory restitution, especially in case of the thumb or index finger, conservation should be attempted.

CRUSHES

The agents which may produce crushing injuries to the hand are almost innumerable. Their size, shape, weight, and consistency, their force and the duration of their impact, create a multiplicity of traumatic lesions. Heat may augment the damage (mangling machines, embossing presses, foundry accidents, etc.). All are characterized by varying degrees of disruption of tissues and devitalization. The insult to soft parts may be only that which accompanies simple fractures, or the hand may be horribly mutilated.

In the simpler cases *x*-rays should be taken at once to determine the presence and extent of bone and joint injury. The circulation in blanched areas of skin may be improved by warm saline packs. Sloughing may ensue. The defect is usually on the dorsum: if small, spontaneous closure may be permitted; if large, grafting should be done (see section on Burns). Subaponeurotic hematomata should be watched and perhaps evacuated. Their organization on the dorsum may occasionally limit closure of the hand; in the base of the palm, they may lead to paresthesia over the distribution of the median nerve of such intensity that grasping of objects cannot be tolerated. In the former group the extensor tendons can be dissected out followed by early motion. In the latter group the annular ligament will need to be divided, the swollen median nerve released and wrapped in fat secured from the base of the palm. Relief is prompt. Fractures should be appropriately treated.

The major cases present a series of problems: shock, pain, circulation, infection, contractures and ankyloses, and finally reconstruction. Shock, if present, must be combated before anesthesia can be given. Only those parts which are hopelessly destroyed should be removed. Fractures should be disregarded, and no fashioning of tissues or even partial closure should be attempted. Dakinization should be instituted and the forearm and hand supported. The extent of circulatory involvement gives anxiety because it cannot at first be estimated. Any evidence of capillary blush in the nail-beds is an encouraging finding. The color and temperature of the hand are unreliable criteria; for parts which are cold and cyanotic may in some hours improve beyond all expectation. Primary amputation at the wrist or amputation of the thumb is, therefore, rarely justifiable. This judgment is sound, even if the general appearance of the hand is frightful, even if there is considerable loss of skin, even if there are multiple fractures, even if sections of tendons are gone and the muscles of the thenar and hypothenar eminences damaged. It is astonishing how much can be salvaged.

While collateral circulation is being developed or lines of demarcation established, infection is to be prevented or treated. To this end dressings must be systematized so that they may be handled efficiently and with a minimum discomfort to the patient. An ingenious retention device which permits ready access to the part will prevent repeated early dressings under an anesthetic. When a part becomes well demarcated, cultures are taken under evipal or gas and the cleansing process is repeated. The gangrenous portion is removed, but no closure is done. Coincident fractures may be moulded into better alignment and dakinization resumed.

The problem of the prevention of ankyloses and contractures is a serious one. Even at this early stage passive motions can be started, despite the fractures. Function is first and always the object of treatment; beautification can come later. The prevention of contractures can best be achieved by skin grafting. If no reconstruction work is to be done, nicely executed Ollier-Thiersch grafts may be placed. If reconstruction work is neces-

sary, and the patient's condition permits, it is best to allow epithelialization to proceed. It may, however, be wise to hasten closure by the use of Reverdin grafts, done under local anesthesia.

After the patient is discharged from the hospital, massage and exercise are carried out for many weeks. On his readmission all scar tissue in the spontaneously closed area (or the whole Reverdin grafted area) is excised and replaced by a skin and fat graft. If only a couple of fingers are involved, either on their volar or dorsal aspects, a tunnel graft may be done. If the palm or dorsum of the hand, as well as the fingers, is concerned, a pedicle graft is done. In about twelve days the pedicle is divided, the graft fashioned and sutured to the remaining margin of the defect. The portions of the grafts which bridge the fingers are cut, fashioned and sutured to the finger defects, usually at several sessions. This is all highly technical surgery—the excision of all scar, the mobilization of the margins of the defect, their gentle handling with tiny hooks (Fig. 1), the avoidance of excess subcutaneous fat, the fashioning of neat finger webs. (Fig. 11.)

The discussion of these details and those of subsequent arthroplasties, bone grafting, osteotomies for adventitious union, fusion of joints in position of optimum function, and tendon grafting or transplantation do not fall within the province of this paper.

CONCLUSION

An attempt has been made to present the treatment of injuries to the hand due to various causes. Certain phases of the subject have been presented with particularity to emphasize the importance of attention to details: systematic preoperative examination based on anatomy and physiology; appropriate instruments, adequate exposure and gentle, deliberate technique to minimize operative trauma; early active motion directed towards the restoration of function, in cases of sutured or grafted tendons accompanied by guarded limitation of antagonistic muscles and in cases of neurorrhaphy accompanied by the employment of devices which permit the use of unaffected muscles but coincidently prevent strain on the nerve suture and on the affected muscles during the reëstablishment of their innervation.

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FUNCTIONING FALSE JOINT OF FINGER FOLLOWING SEVERE TRAUMA

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Introduction. Severe injuries of the hand are often not so serious in nature as they appear on first examination. Recently more emphasis has been placed on conservative treatment of traumatized hands. Such injuries are too often followed by hurried amputations, whereas the initial treatment frequently should stress the promotion of circulation and prevention of infection. It is wise to adopt a conservative attitude towards a traumatized member, even though the chance of maintaining circulation appears small. Asepsis can be maintained by having instruments, sutures and sterile gauze ready for use. An example of this type of treatment is given in the following case report:

Mr. W. W., an engineer, age 47, was operating a circular saw on March 31, 1938. His hand slipped and the saw cut his right thumb and index finger.

Examination. The patient was bleeding profusely from the hand, with the thumb and index finger hanging by flaps of skin on the palmar surface. The thumb was sawed from the first metacarpal and first phalanx obliquely to the second phalanx. The second phalanx of the thumb was dislocated, and the soft tissues and tendons, including the flexor and extensor pollicis, were cut and lacerated.

The right index finger was also cut through, except for a small flap of skin on the flexor surface. Most of the proximal phalanx was completely torn out by the saw. The extensor and flexor tendons were severed and the soft tissues macerated.

There was no function of either the thumb or the index finger. The remaining stump of the proximal phalanx of the index finger functioned, due to the fact that the first lumbricale and interossei muscles are inserted into the base of the first phalanx and into the aponeurosis of the common extensor tendon. The circulation of both members was apparently not impaired.

Operation. The unusual feature of this operation was that it was done under adverse circumstances. The patient, being an engineer, would not leave his post of duty after the accident, and therefore the first aid operation was done in the engine room with poor light and no assistance.

Naphtha and ether were used to cleanse the hand, followed by tincture of metaphen. One per cent solution of novocaine was used as a local anesthesia.

The right index finger was treated first by approximating the middle phalanx as closely as possible to the remaining fragment of the proximal phalanx. The severed flexor and extensor tendons were sutured with through-and-through sutures and one long mattress suture of catgut. Hemorrhage was controlled by ligation of the vessels and also by a rubber tube tourniquet around the forearm. The aponeurosis and subcutaneous tissue were drawn over the tendons with a fine catgut and the skin closed with dermal sutures.

About the same procedure was followed with the thumb, in addition to the reduction of the dislocation of the first phalanx and the approximation of the fragments of the first phalanx.

The hand was placed in a plaster of Paris splint with the thumb and index finger in extension. Anti-tetanic serum was administered. Daily dressings were necessary for the first few days until the danger of infection and gangrene from poor circulation was past. Reliance for maintenance of circulation was in the blood supply from the flap of skin on the palmar surface. If infection did not set in and the blood supply was sufficient, the patient would have the benefit of the conservative treatment; if not, an amputation could be made at a later time. Fortunately no infection took place. There was some sloughing of the tissues on the dorsal surface of the index finger, and later the extrusion of an unabsorbed suture.

Progress. With each dressing the circulation of the thumb and especially that of the index

finger appeared good. The aim of the operation was to bring about union between the middle phalanx and the remaining stump of the remaining portion of the first phalanx and the mid-phalanx. (Fig. 1.)

Vasomotor and atrophic changes with edema

FIG. 1. *X*-ray taken two months after accident, showing artificial joint formed between remaining portion of first phalanx and middle phalanx of index finger. Note absence of distal two-thirds of the first phalanx and resulting foreshortening of the index finger. The comminuted fracture of the proximal phalanx of the thumb has healed with good position and union.

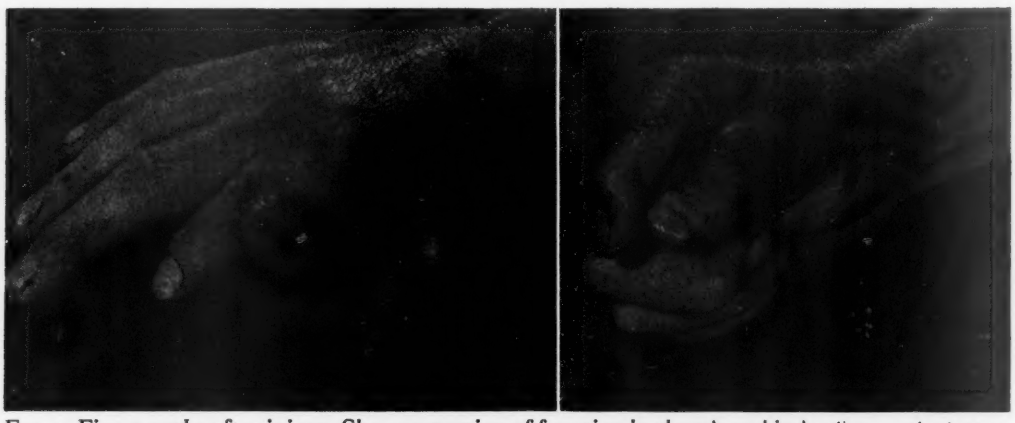

FIG. 2. Five months after injury. Shows retention of function in thumb and index finger; the latter, in spite of its abbreviation, remains normal in every respect. No perceptible atrophy has occurred. Blood and nerve supply to fingers and finger-nail beds remains good.

proximal phalanx of the index finger. The injured man would then have a finger with two phalanges instead of three. However, recent *x*-rays taken by Dr. James M. Flynn show an artificial joint has been formed between the from long immobilization were prevented by early massage and passive movements.

The patient resumed his regular occupation June 13, 1938, after a period of ten weeks. He has at least 90 per cent of normal thumb func-

tion at the present time. He can flex, extend, adduct and abduct the index finger, but is not able as yet to flex the distal phalanx. The right index finger is shorter than that of the left hand, reaching to about the level of the middle phalanx of the second finger, but it is in good alignment. The circulation is good, the fingernail is healthy, and there has been no apparent atrophy either of the thumb or of the index finger. The patient is following his regular work at the present time with not much disability.

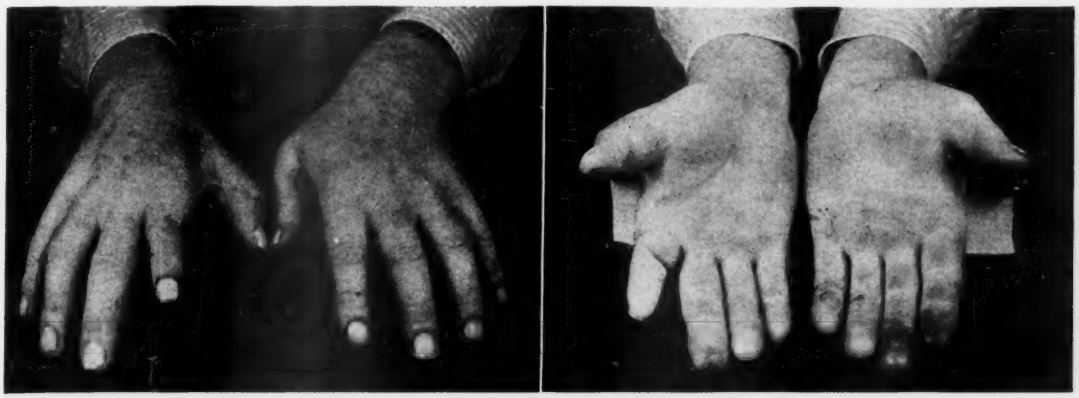

Fig. 3. Comparative view of both hands, taken at same time as Figure 2. Note shortening of right index finger, but completely normal appearance of hand.

CONCLUSION

The successful result in this case is attributed to the fact that the remaining circulation following injury was not impaired, and the operative and postoperative care was carried out to maintain and promote circulation and prevention of infection. The formation of the false joint of the index finger is attributed to keeping in position the remaining portion of the proximal phalanx and the mid-phalanx by uniting the tendons and the aponeurosis of the common extensor tendon.

In the presence of working conditions in the engine room, it would have been unwise to carry out any other type of surgical procedure. The aim was to save the thumb and index finger, thus making a useful hand without much disfigurement. (Figs. 2 and 3.)

FRACTURES OF THE UPPER EXTREMITY OF THE FEMUR—NECK AND TROCHANTER

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THE subject of surgery of fractures of the trochanter and neck of the femur, it would seem, should first resolve itself into the proper choice of method to be used by the individual surgeon, giving due consideration to the capabilities in surgery and the physical advantages available for handling of the case. The use of any operative procedure, to offer the best expectation of success, should be in the hands of surgical service well qualified and of course entails hospitalization. This cannot under all conditions be available, but at the present offers little objection due to the improvement in adequate facilities for transportation and the ever increasing availability of hospitals and trained surgical personnel.

CHOICE OF METHOD

Fractures of the neck of the femur can be adequately reduced by traction, but it is extremely doubtful if they can be properly maintained in desired immobility in this way. As complete immobility is necessary for union in this type of case, reduction by traction should be followed by the application of a cast to provide the completest possible immobility. Intertrochanteric fractures do not present quite the same problem, nor do they demand such absolute immobilization. These have been successfully reduced and maintained adequately until union has taken place by the use of traction alone.

In both instances, that is in fractures of the neck and in intertrochanteric fractures, many variations in traction application have appeared so that something must be said covering this phase. For those cases under consideration it has not been found necessary in any instance to resort to skeletal traction in any form. Adhesive traction applied according to the Russell method has proved entirely satisfactory, if properly done with appreciation of the physical principles involved. Various individual modifications, in my experience, tend to reduce rather than increase its efficiency. The reader is referred to Russell's original articles for proper description.

Little need be said here concerning the Whitman method of reduction or abduction cast as this is generally familiar and no doubt is the method most frequently used where internal fixation is not feasible or desirable. Since publication of the Leadbetter method of reduction, this procedure is now used to reduce the fragments before application of the cast for immobilization. Results published show a high incidence of success. The use of such procedures must not be entirely thrown into the discard as a result of the present popularity of internal fixation. No matter how much one may be convinced of the superiority of internal fixation by one method or another, it must be realized that there are those in whose hands it is not possible. Also, some patients, though not many, will refuse operation, but should still be offered some method with reasonable assurance of success. Internal fixation should, of course, not be considered under conditions where proper hospital and trained surgical facilities are not at hand. It is under these conditions that I feel reduction and immobilization in a cast are indicated. Published statistics, however, show a higher incidence of union where internal fixation is used, the percentage ranging from 80 to 95.

INTERNAL FIXATION

My belief is that internal fixation is the method of choice in those cases in which it is applicable and these certainly constitute the greater majority of those sustaining an injury of this type. Fractures with comminution which involves the head, neck, or trochanter cannot be fixed and held by internal fixation, but these are comparatively rare. It is fortunate indeed that the great majority of neck fractures are transverse or nearly so and hence lend themselves to immobilization by internal fixation. Of all, the subcapital fractures offer the smallest chance of success as it is difficult to get sufficient purchase of that part of the fixing agent in the small head fragment. In addition, the shorter the head fragment the less the possibility of an adequate circulation.

Fractures of the neck proper may be successfully held by internal fixation; assurance of union increases in direct proportion to the length of the capital fragment. Intertrochanteric fractures are likewise successfully handled by internal fixation so long as the great trochanter or the major portion of it remains as part of the shaft fragment. If the fracture line is so placed as to sever the great trochanter from the shaft it is doubtful if the projecting portion of cortex of the shaft will be sufficient to hold the fixing agent without refracture.

I would stress here the importance of the complete immobility of the fixing agent in bone, no matter for what type of fracture it may be used. This is of prime importance, for it must be realized that where any motion is allowed, the fixing agent, be it screw, wire, nail, bone, or material of special metallic content, will become a foreign body and cause certain failure. Proper attention to this in the operative technique I believe to be of greater importance than the choice of some agent of special composition, although recently the work of Venable and Stuck has thrown a somewhat different light on this problem. Of course, it is supposed that asepsis must be complete and absolute. Much has appeared recently concerning special alloys and materials, but experience leads me to believe that the solution of the problem depends more on proper asepsis, proper technique of application, and resultant absolute immobility of the foreign material in the bone.

The majority of cases lend themselves to internal fixation. The advantages of this method far outweigh the general disadvantages usually held to exist in surgical procedures on individuals in the upper age brackets. The procedure used according to the Martin method has been well tolerated in all cases and in itself constitutes far less danger to the patient than would continued confinement in the recumbent position.

At this writing there seems to be a divergence of opinion as to the proper time for operation, some advocating immediate reduction and others gradual reduction with application of internal fixation at some period, usually during the first two weeks. Most upper femoral fractures occur in the aged and more or less infirm, and a nervous and physical cataclysm usually is an immediate result of the accident. This has already taxed the diminished reserves to capacity, and therefore it hardly seems wise to add the additional trauma and shock of manipulation, operative procedure, and anesthetic at this time. Judging by results obtained, a delay of a few days (five to ten) for gradual replacement by traction has in no wise affected the ultimate outcome. The patient is in better physical condition, initial shock has been overcome, nervous adjustment has taken place, and it is believed that local issue resistance is increased by healing of soft parts injured at the time of fracture.

In the procedure of internal fixation, with its various recent modifications, a wide range of fixing agents and incisions have been reported. At present most authors stress simplification and extra-articular introduction, while little is now seen of the truly formidable open reduction with exposure of the hip joint and fracture

site. The essentials of the present simplified technique of preoperative reduction, simple lateral incision and extra-articular fixation with maintenance of proper position during the procedure, were stressed by Martin as early as 1920. It is altogether fitting to give full credit and priority to this pioneer in the field of modern internal fixation. This work antedates the presently popular methods by some years and has yet to be supplanted by any procedure or fixing agent which is simpler or more efficient.

TYPE OF FIXING AGENT

In the selection of the type of fixing agent to be used the first essential is sufficient strength to withstand the shearing force of muscle pull at the site of fracture. The substance chosen must also maintain position, prevent rotation, hold the fragment in close apposition, and at the same time provide only a minimum of interference with blood supply and normal physiology of bone regeneration and union.

Many of the appliances now in use fulfil in some way most of these requirements. Units such as single bolts, wires, pins and nails may satisfy the need for strength, but it is not inconceivable that these can, and at times do, act as axes of rotation. This is particularly so if the appliance is of the smooth type depending on friction and muscle pull alone to maintain close apposition at the fracture site. The use of multiple (two or more) fixing agents or of a flanged nail of course overcomes this, but here again friction and muscle pull are alone depended upon to give continued close apposition. In some instances there is also forceful impaction created by the driving force applied in inserting the appliance or in "driving it home." In such cases, although I do not recall having seen it reported in relation to internal fixation, the danger of fat embolism and the effect of trauma in producing it cannot be entirely dismissed.

With relation to minimum interference with blood supply, the small wire or pin of course creates the least displacement or replacement of tissue. Larger appliances displace more bone and hence must infringe upon the vascular bed in the cancellous structure of the neck and head. The flanged nail type, although of little actual metallic area in its cross section, divides that portion of bone through which it passes into three compartments and must of necessity divide many blood vascular channels which otherwise would communicate between these compartments. Thus there results some measure of interference with the blood supply of an area equal to the circumference of the nail throughout its length.

The double screw application seems to meet most of the above mentioned demands for a simple and efficient appliance. Sufficient strength is provided and prevention of rotation is accomplished by the use of two screws. Friction and muscle pull are not relied upon to maintain position as there exists definite tightening action across the fracture site by the pull of the threads engaged in the head fragment, causing mild, though forceful, apposition of the fragments. In addition, replacement of tissue by metal and interference with circulation, although not at an actual minimum, are not excessive.

In this procedure the fixing agent used is the ordinary carpenter or hardware screw No. 10, usually 3 inches in length, which has previously been case hardened. Formerly the No. 8 screw had been in use but recently the No. 10 has been adopted as it seems to give a better purchase when the threads engage the head fragment. There has been no difference noted in cases in which the different sized screws were used, so this change is probably optional. So far as simplicity is concerned this method requires no special instruments for its proper application. With serial *x*-ray check as advocated, it has been found possible to introduce the screws with sufficient accuracy. Aside from the usual operating tray, one requires only the screws and a drill point of proper size. For the No.

8 screw a 5/32 drill is used and for the No. 10 a 3/16.

TECHNIQUE

After hospitalization and *x*-ray study (Fig. 1A) Russell traction is applied until gradual replacement is secured as demonstrated by subsequent portable *x*-ray. In only one instance has traction failed to produce proper reduction, necessitating a resort to the Leadbetter method of reduction under anesthesia on the operating table.

When proper replacement has been demonstrated by *x*-ray, usually in five to seven days, the patient is taken to the operating room. Traction has overcome muscle tonus so that in transporting the patient it has been found necessary only to guard against external rotation. Previous deformity or displacement does not recur during this period as long as external rotation has been prevented. With the patient on the table and the anesthetic of choice administered, the foot on the injured side is held by an assistant, with sufficient traction to maintain length and with the leg in a position of slight abduction and internal rotation of about 30 degrees. At this time, before actual operative procedure has begun, a portable *x*-ray is used to verify position. (Fig. 2A.) Serial *x*-ray checks are facilitated by placing the patient on a tunnel cassette so situated as to make it possible to introduce and remove plates on the side of the table away from the operative field.

With the most recent plate in view, simple lateral incision is made with the prominence of the trochanter in the upper angle of wound. The incision is carried directly down to bone, exposing the area beneath the trochanter and the upper portion of the shaft. This area between shaft proper and prominence of the trochanter will usually be at a right angle to the long axis of the neck. Any slight variation noted in the *x*-ray may be easily compensated for. A point in this plane on the prominence of the anteroposterior curvature of the cortex is chosen and the drill introduced parallel to the long axis of the neck. It must be remembered that

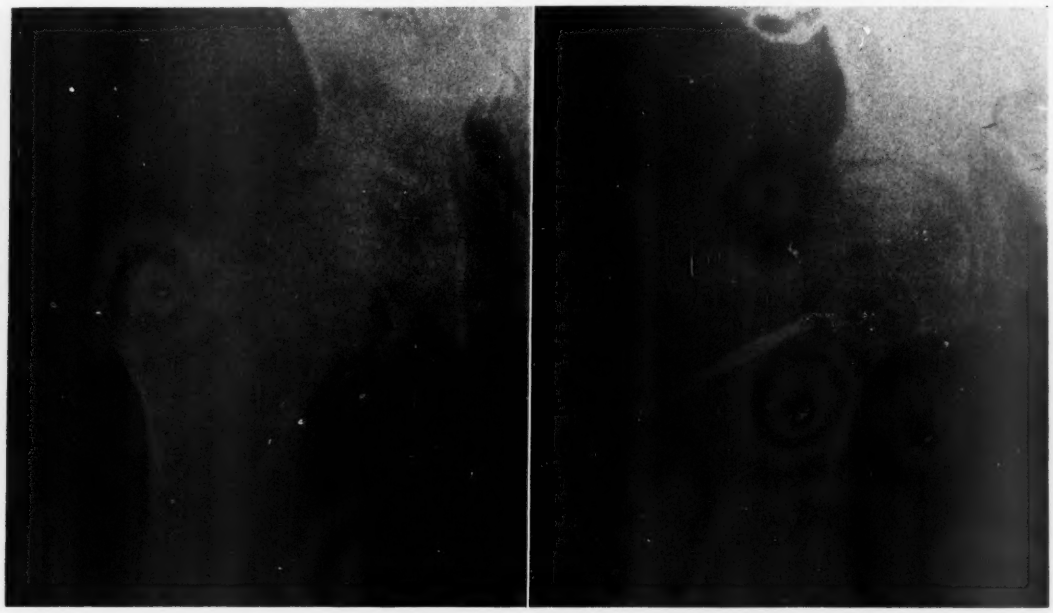
Fig. 1. *X*-ray on admission.

the neck of the femur is normally slightly (10 to 15 degrees) inclined forward or anteriorly, and this must be allowed for in introducing drill and screw. The drill is introduced approximately *to* the line of fracture. The head fragment is not drilled. An *x*-ray is made with drill in place to verify proper direction. (Fig. 2B.) If not satisfactory, the drill may be redirected without harm.

The drill is then removed and the first screw inserted and partially screwed in. The projecting portion serves as a guide by which the second screw is placed in a similar manner adjacent and parallel to it. Both screws are then "driven home," firmly fixing the fracture site. A squeezing action is produced as the screws when tightened pull the head fragment toward the shaft portion of the neck. With the two screws in place, final *x*-ray check is made (Fig. 3A) in both anteroposterior and lateral views. The lateral projection is made by flexing the thigh to a right angle with the plate beneath the buttocks. (Fig. 3B.)

Mild traction and suspension for a period of seven to ten days postoperatively

add a feeling of security. This is optional, however, as there have been cases in which complete freedom of motion was permitted being absolute avoidance of weight bearing for a period of six months or until *x*-ray demonstrates union.



A B

FIG. 2. A, complete reduction (Leadbetter manipulation). Portable *x*-ray on table immediately preoperative. B, portable intermediate picture with drill in place. Screw was redirected slightly upward.

A B

FIG. 3. A, both screws in place. B, lateral view, made by flexing thigh to right angle.

immediately following operation. If postoperative traction is used, it is usually discontinued when the sutures are removed. Complete freedom of motion is then allowed and the patient is encouraged in activity. Rolling chair, then crutches, are added, the only restriction insisted upon

RESULTS

Since femoral fractures of this type are, with few exceptions, sustained by the aged, I believe that some explanation of what is meant by a "successful case" must be offered. Life expectancy in most of these

individuals is nil, or almost so. Consequently there will always be some patients who will not survive the long period of convalescence, but will die of natural causes unrelated to the injury or procedure. If these patients have had a successful application of internal fixation and thereby been rendered completely comfortable and active with the exception of actual weight bearing, I do not feel that they can be listed among the failures, although union may not be present at the time of death. Many statistical studies report bony union in 80 to 90 per cent of cases in which

A

B

Fig. 4. A, operated February 1929. Screws in place nine years and two months. B, left hip operated January 1932. Screws in place six years and five months. Right hip fractured April 1938. Vitalium screws.

internal fixation has been used, so that, if such "successful cases" are included, the percentage of satisfactory results will approach as near perfection as we can reasonably hope to attain—certainly more so than by any other means at hand.

SUMMARY

Internal fixation appears to me the method of choice in dealing with fractures of the neck of the femur and intertrochanteric fractures. Of the many methods advanced, the Martin two-screw application is preferred from the standpoint of simplicity and efficiency. It gives results comparable to those reported by the use of procedures more popular at present.

To date the cases forming the basis of this presentation are eighty in number and in the hands of twelve surgeons. This attests to the possibility of general use of the procedure by men qualified in surgery though possessing no special qualifications in the field of bone work.

My thanks are hereby expressed to those who have so kindly allowed consideration of their cases in this presentation. Also to Dr. J. C. Rodick and the staff of the *x*-ray Department of Touro Infirmary, and to Drs. Samuel and Bowie for their kindness and assistance in procuring *x*-rays.

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The typical picture [in fractures of the neck of the femur] is that of an elderly woman lying in bed complaining of pain in her hip and of inability to move the leg following a slight fall in her home or on the street.

INTERNAL DERANGEMENT OF THE KNEE JOINT

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IN this discussion of internal derangements of the knee joint we shall limit ourselves to intra-articular lesions, and principally to those lesions which are the result of primary and secondary trauma. Mentioned in frequency of occurrence they are:

1. Injury and pathologic processes of the semilunar cartilages.
2. Injury to the crucial and lateral ligaments.
3. Loose bodies.
4. Traumatic lesions of the synovial membrane and masses.

Inasmuch as injuries to the *semilunar cartilage* represent the largest percentage of internal derangements of the knee, these will be given first consideration. Approximately 90 per cent of cartilage injuries are of the internal meniscus, and these occur much more frequently in the male than in the female. However, the ratio is becoming less as the modern woman becomes more athletic and exposes herself to this common athletic injury. The mechanism and cause are simple if one studies the forces and position of the leg when the injury occurs and applies this knowledge to the principal anatomic structures that are affected. The vast majority of injuries to the internal semilunar cartilage are produced when the thigh is in an adducted position, or when the lower leg is in an abducted position and the knee is flexed to about 30 to 45 degrees, or when the knee is in extreme flexion and the thigh is forced forward. For example, in the first instance, a football player or other athlete is running and suddenly attempts to go into a position at an angle which adducts the thigh while the foot and leg remain in the horizontal position. The result is that a sudden side and twisting strain is thrown upon the ligaments on the inner aspect of the knee joint and these ligaments become stretched or torn, more often the former. When the leg is fully extended, the internal lateral ligament is in its most advantageous and strongest position and one, therefore, seldom sees an injury to the internal lateral or crucial ligament that has been produced by a blow to the outside of the leg when it is in full extension.

The semilunar cartilages are attached to the internal and external lateral ligaments, and their anterior and posterior tips are firmly fixed to the tibia by strong independent ligamentous attachments. It can be readily understood how a blow or twist to a flexed leg that forces the knee toward the midline of the body could cause a loosening of the cartilage at its lateral attachment because of the firm fixation of the anterior and posterior tips of the cartilage to the tibia. If the leg is acutely flexed the rent will more often start in the anterior portion of the cartilage, as is seen in occupational injuries where it is necessary for the worker to assume a kneeling position, as miners, carpenters, etc.

A very slight twist of the leg will often cause the first attack. The patient will usually give a history of having had a sudden wrench or twist of the knee or, as he will describe it "his knee slipped out of joint," and the knee is momentarily or permanently held in a locked flexed position with an inability to extend the leg. Slight manipulations by the patient or by a companion are often resorted to and the cartilage reduced, and on examination the patient will present more or less typical signs: (1) very rapid development of effusion in the knee joint with point

sensitiveness about the anterior portion of the internal semilunar cartilage; (2) pain about the inner aspect of the joint

Fig. 1. Fig. 2.

Fig. 1. Jones knee cage, front view.
Fig. 2. Side view.

when an attempt is made to extend the knee if the cartilage is still out of position. If the external semilunar cartilage happens to be the damaged one, then, of course, the point sensitiveness will be on the outer side of the knee joint and the patient will identify this location as the site of his discomfort.

Immediate treatment is very important. Where full extension cannot be obtained manipulation and reduction without anesthetic should be attempted. One can usually accomplish this by flexing the leg and, supporting the knee with one hand, internally and externally rotating the leg, and then quickly extending the knee with the leg in the position of internal rotation. A snap is quite often felt and then the patient is able fully to extend the leg. However, if the rent is sufficient to displace the cartilage beyond the midline of the condyle of the femur it may be irreducible and forced extension of the leg will tend to increase the tear in the cartilage. In our opinion this seldom occurs if reduction is attempted without anesthesia. Further treatment is based upon definite clinical and pathologic facts. An important point to be remembered is that a rent in the body of the cartilaginous

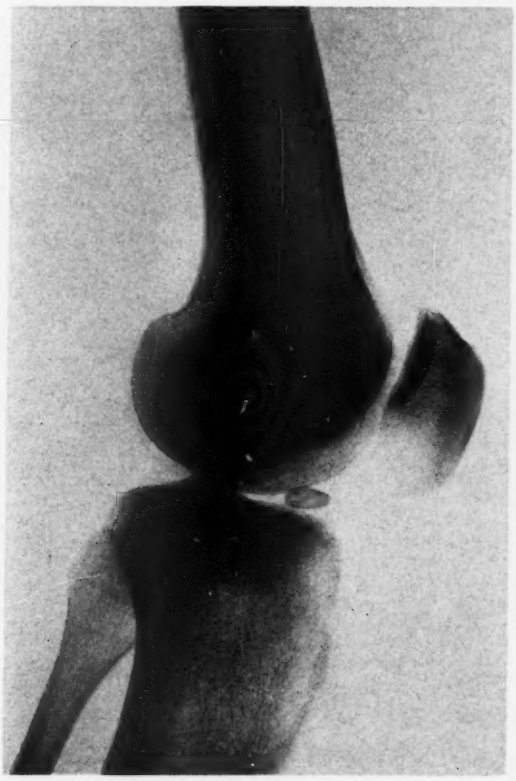

Fig. 3. The usual type of loose body in the lower compartment of the joint which, at times, becomes wedged between the crucial ligaments.

portion of the meniscus will not heal; if the rent occurs in the periphery close to the attachment of the lateral ligaments, at the point where there are blood vessels and fibrous tissue, healing may take place. It is impossible to ascertain from the physical examination alone the exact location of the rent. For this reason, in the absence of permanent locking of a cartilage that is irreducible, we feel one should give the patient the benefit of the doubt and place the leg in a well fitting plaster or crinoline dressing in a straight position for sufficient time to allow healing to occur, which is approximately four weeks. If, after active use, physiotherapy and development of the muscle tissues, the patient suffers a second attack then operation is indicated and the postponement of operation is not advisable. While repeated

attacks of locking of the knee seldom add to the magnitude of the operation or militate against a good end result, however, to permit exploration of the knee so that one can be sure of his diagnosis before proceeding further with the operation.

A B

FIG. 4. Unusually large but typical osteochondritis dissecans. A, anteroposterior view. B, lateral view.

a fall caused by sudden locking of the knee may result in other serious bodily injury. I, personally, have seen two fractured skulls and several cases of major injury caused by locking of the cartilage when the patient was descending the stairs.

There is no operation of the knee joint that is more satisfactory than the removal of a semilunar cartilage when not associated with complications which may be the causative factor, such as fracture, etc. The technique of the operation has been described so many times that it hardly seems necessary to discuss it here in detail. The author prefers a small incision running obliquely across the knee, approximately 1 inch medial to the patella tendon and extending outward over the upper section of the internal condyle. The exposure through the ligamentous tissues is usually not over 1 inch in length, just long enough A cartilage in which a rent cannot be demonstrated is not removed. This can usually be determined by introducing a small blunt hook and passing it around the margin of the cartilage. In some cases it is difficult to demonstrate a tear in the posterior portion of the cartilage. When the diagnosis of the suspected cartilage is made the incision of the capsule may be enlarged, if necessary, to facilitate its clean dissection.

The postoperative treatment consists of immobilization in a crinoline dressing for a period of two weeks. A soft dressing is then applied and the patient is allowed to use the knee joint and develop the musculature of the leg, which plays such an important rôle in the stability of the knee. The average athlete under twenty-five years of age is usually able to return to an active athletic life six or eight weeks

after operation, earlier in some instances if the cartilage injury was not associated with stretching or rupture of the internal lateral or anterior crucial ligaments.

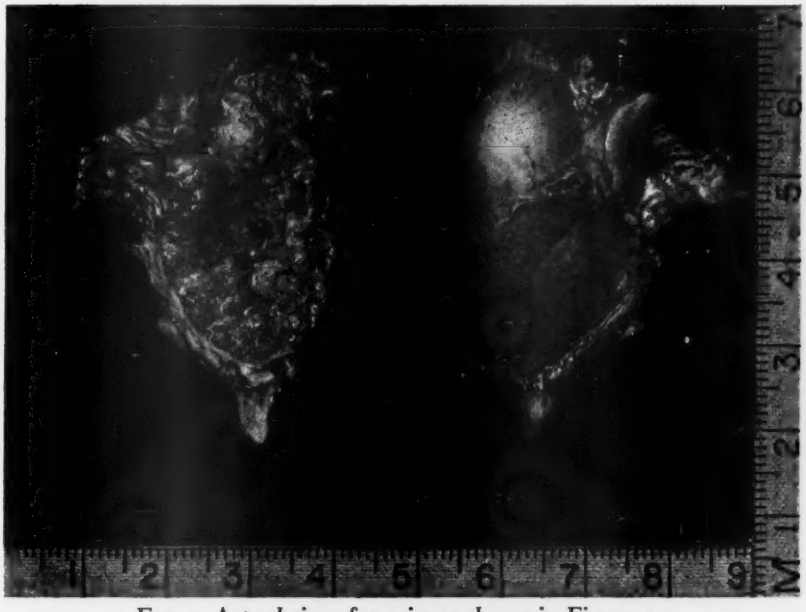

Fig. 5. Actual size of specimen shown in Figure 4.

In 25 per cent of cases of injury to the internal semilunar cartilage we have found sufficient relaxation of the internal lateral ligament and of the fascia on the inner border of the knee, as well as relaxation of the crucials, to justify consideration. The symptoms of a relaxed knee so closely simulate those of an injury to the cartilage that one has a great deal of difficulty in differentiation. The pseudo-locking or momentary locking of a relaxed knee is often misleading and the removal of an internal semilunar cartilage that shows no pathology will not rectify the relaxation which is causing the patient's symptoms. One must decide at the time of operation for the removal of a definitely abnormal cartilage whether there is sufficient relaxation of the knee joint to justify a more extensive operation for the correction of this associated condition. In the absence of any demonstrable evidence of a tear in the cartilage, it will be found that the operation of reinforcement will be sufficient in many cases to relieve the patient of his symptoms. A number of procedures have been described for the correction of this condition. For many years I have been doing an overlapping plication of fascia and ligamentous tissue on the inner border of the knee joint and a reinforcement of the inner aspect of the knee joint by means of fascial sutures and strips taken from the tensor fascia lata. This procedure has been very satisfactory in our hands. When this operation is done the immobilization period of four weeks is followed by physiotherapy and active use. The convalescent period, of course, extends over a longer period of time. The point I wish to stress, if one expects a satisfactory result, is—do not remove a cartilage that shows no pathology and do not overlook relaxation of the knee which is associated with approximately 25 per cent of injuries to the internal semilunar cartilage.

Injury to the Crucial and Lateral Ligaments. Injury to the crucial and lateral ligaments must necessarily be divided into two general groups: (1) Moderate relaxation of the knee joint which allows slight forward or backward sliding of the tibia upon the femur; or a slight increase of lateral mobility which may be the result of stretched or torn lateral ligaments and

fascia plus a similar condition in the crucial ligaments. (2) Severe relaxation of the knee, or the so-called rocker-knee, in which the tibia slides forward on the femur when there is a tear in the anterior crucial, and backward on the femur when the tear is in the posterior crucial ligament. Associated with this is a very marked lateral mobility, so that the lower leg can be put in either a bow-leg or knock-knee position.

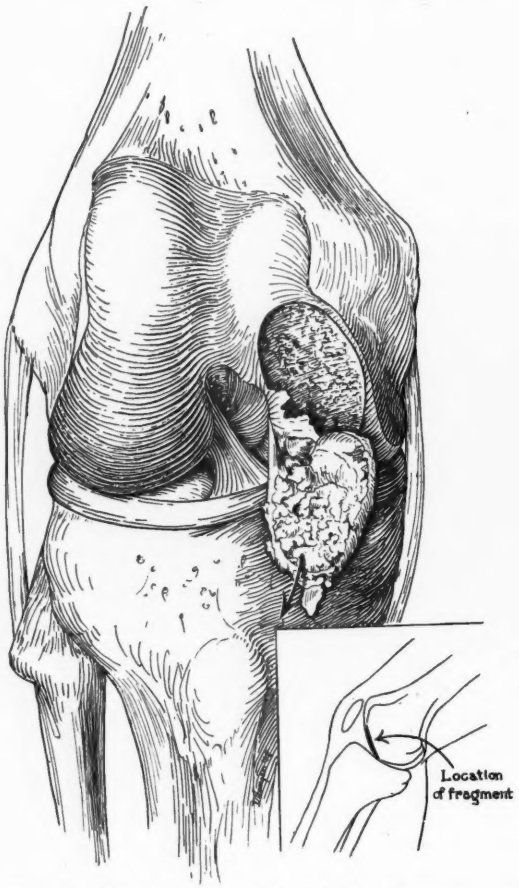

FIG. 6. Sketch of osteochondritis dissecans of unusual size shown in Figure 5.

Obviously, these two conditions require entirely different modes of treatment. The latter condition, or the severe relaxation, can be avoided in many cases by early treatment of a rupture of the lateral ligaments. In instances of uncomplicated rupture from severe trauma an immediate suture is indicated followed by a period of rest over a minimum of four weeks. Repair of a torn crucial ligament cannot be accomplished by suture, and inasmuch as the knee joint depends on the lateral rather than the crucial ligaments for its stability it seems that these should be given the major consideration.

I should like to discuss as concisely as possible the treatment of these chronic relaxations. In our discussion of injuries to the semilunar cartilage we called attention to the milder form of relaxation most often associated with injuries to the internal semilunar cartilage and we stated that an injury to the anterior crucial ligament or the internal lateral ligament is caused by the same type of strain which produces an injury to the cartilage. However, there are instances in which there is no injury to the cartilage and an individual who leads an active athletic or industrial life where agility and strength are required, is inconvenienced and seriously handicapped by an instable knee which, at times, pseudo-locks because of excess rotation and the slipping of the tibia upon the femur. The use of a Jones knee cage will very often enable such an individual to carry on an active life.

When an individual does not wish or refuses, to wear the knee cage, then the operation of fascial reinforcement and stabilization by means of fascia about the knee joint is very satisfactory. However, in the extreme rocker knee, where there is a very marked relaxation of the knee joint with stretching or tearing of the crucial and lateral ligaments, the conservative type of operation will prove of no avail. In this patient one must make an effort to stabilize the knee by one of the more radical intra-articular procedures, many of which have been described by Groves, Krida, Carrell and others. The principle of these procedures is the manufacturing of crucial ligaments by means of tendon and fascia. Mauck has recently described a procedure in which he transplants the insertion of the fascia and the internal or external lateral ligaments of the knee to a lower level on the tibia. In extreme and borderline cases this is a very satisfactory operation and one which is

not so extensive as restoration of the crucials by means of tendon and fascia.

Loose Bodies. Loose bodies are of two general types: the isolated loose body which is the result of trauma; and multiple loose bodies which are the result of abnormal cartilaginous growth, may be secondary to repeated trauma, and occur in the rather young individual during the growth period or in the latter period of life secondary to hypertrophic arthritic changes. The traumatic loose body, which is usually the isolated or single body, is more often the result of industrial injury than are the multiple loose bodies. For several months after a very slight trauma to the patella, tibia or femur, it is not unusual for a patient to complain of partial or complete locking of the knee, and examination will reveal evidence of irritation in the quadriceps pouch or about the lateral borders of the knee joint with effusion. The patient will very often make his own diagnosis and will tell you that he feels something slip in his knee joint. Upon careful and thorough examination one is able to palpate the loose body which is the cause of the discomfort. If the *x*-rays of such a knee are scrutinized it will be found that at the time of injury a small piece of bone and cartilage was loosened and had become sufficiently hypertrophied to produce symptoms. (Fig. 3.)

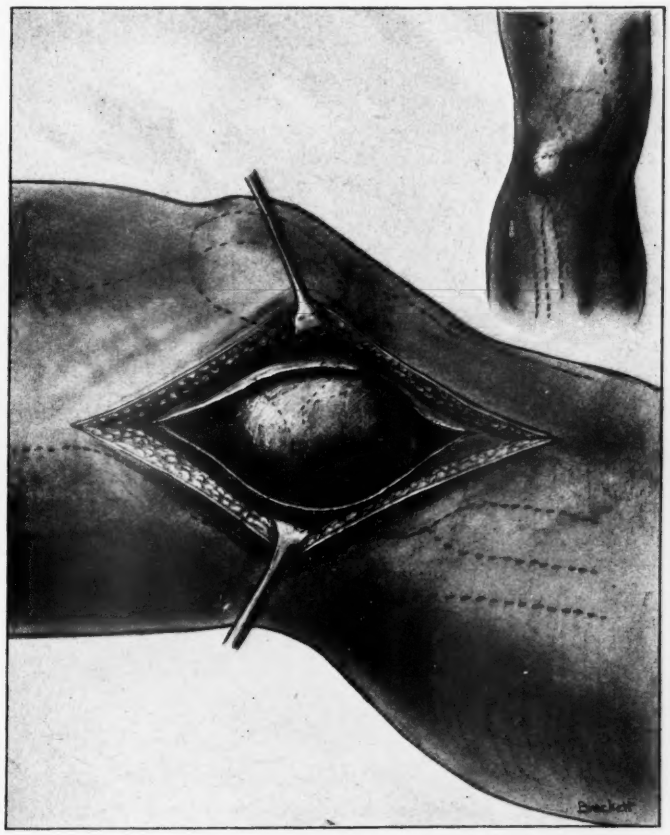

Fig. 7. Very large cyst of subpatella fat pad.

Multiple loose bodies which are the result of abnormal growth and proliferation, either from the tibia, femur or patella, are very often seen in patients ranging from 10 to 15 years of age. On exploration of the knee joint for removal of these loose bodies, one may see about the inferior border of the patella an area of rough, corrugated hyperplasia from which small bits of cartilage have been flaked. Shands, in his experimental work, proved very clearly that trauma will produce hypertrophy of hyaline cartilage. Therefore, one

can see why loose bodies which are the result of this condition should arise more frequently from the patella than from the

Fig. 8. Drawing of section of cyst.

other bones entering into the knee joint, as the patella is subject to more strain and trauma than the other bones.

The third type of loose body is the osteoarthritic type, excluding the so-called osteochondromatosis, and is the result of proliferation along the margins of the tibia, femur and patella. It is probable that these portions of cartilage are broken from their host and undergo a gradual increase in size. This type of loose body is frequently multiple in character.

Osteochondritis dissecans, which is not an infrequent cause of loose bodies, demands special consideration. This peculiar pathologic process, regarding whose etiology there are several hypotheses, is a curious one. It has never been definitely proved whether a quiet aseptic necrosis occurred from obstruction to the circulation, non-traumatic, or whether the aseptic necrosis was secondary to a traumatic lesion. With or without a history of injury, the patient begins to complain of discomfort and slight locking or catching of the knee, and the *x*-ray will show a very definite hiatus on the inner border of the internal condyle of the femur. As the *x*-ray findings are typical, diagnosis is relatively simple. Upon exploration it will be found that this cartilage may be partially attached and dangling, producing the sensation of locking. The flaking off of pieces of cartilage and osseous tissue which undergo proliferation is another etiologic factor in the production of loose bodies. Operation for the removal of semi-attached or completely detached loose bodies followed by a period of rest gives a very gratifying end result. It has been the author's experience that permanent locking of the knee joint is often caused by the impingement of the loose body between the crucial ligaments so tightly that it cannot be dislodged by ordinary manipulation.

The operative removal of loose bodies may be a very simple procedure or the surgeon may encounter considerable difficulty. There are one or two practical points that it may not be amiss to mention here. If it is possible to manipulate a single loose body into a position about the lateral borders of the knee joint so that a heavy skin needle can be inserted through the skin and into the loose body, anchoring it so that it does not move from its position, any loose body can be removed through a very small incision. If a large incision is necessary for the elusive type of loose body, manipulation and milking of the knee very often will bring the loose body in situ and it can be easily removed. From time to time one sees loose bodies that are partially encysted in synovial sacs or are partially attached to the synovial membrane. However, the removal of loose bodies is a very satisfactory procedure. In those which are caused by proliferation in the growing child or as a result of osteoarthritis, there is a tendency to recurrence; in the child this may be true during the entire growth period.

Injury to the Synovial Masses and Synovial Tissue. The most frequent injury is that of the infrapatella pad of fat and synovial tissue which lies beneath the patella tendon and is a combination of synovial folds and fat. We shall not discuss the details of its histologic structure, but will mention only the practical points. This pad has its so-called alar ligaments, which are synovial folds and attach to the

intercondylar notch of the femur anterior to the crucial ligaments. They serve to suspend the mass which is broad at its base and reflected to the anterior extremities of the semilunar cartilages and attached to the tibia. When the knee is in a fully extended position the pad is most prominent at the lateral border of the patella tendon. This mass of tissue, of course, is subject to inflammatory disease as is any tissue that is composed of lipomatous and synovial tissue. We will not take up the question of inflammatory changes, such as are evident in arthritic and tuberculous conditions, but will confine our discussion to the single traumatic lesion and those lesions which are secondary to repeated injury. The location of the subpatella fat pad is such that it is often subjected to direct trauma by falling when the knee is in a flexed position, or by direct blows, with the result that hemorrhagic

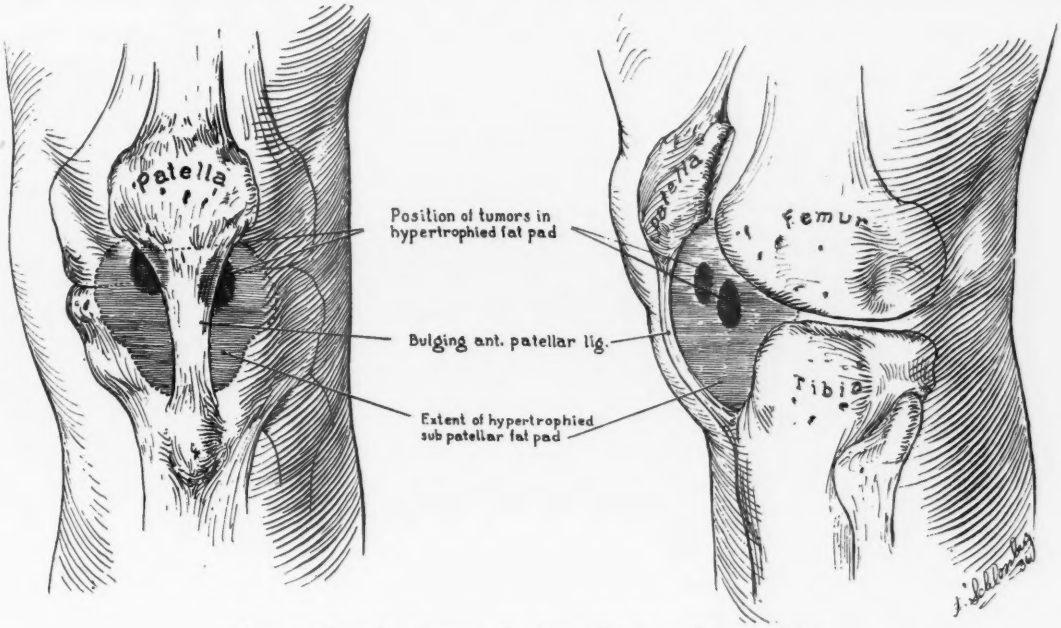

Fig. 9. Fibrochondroma of subpatella fat pad—multiple.

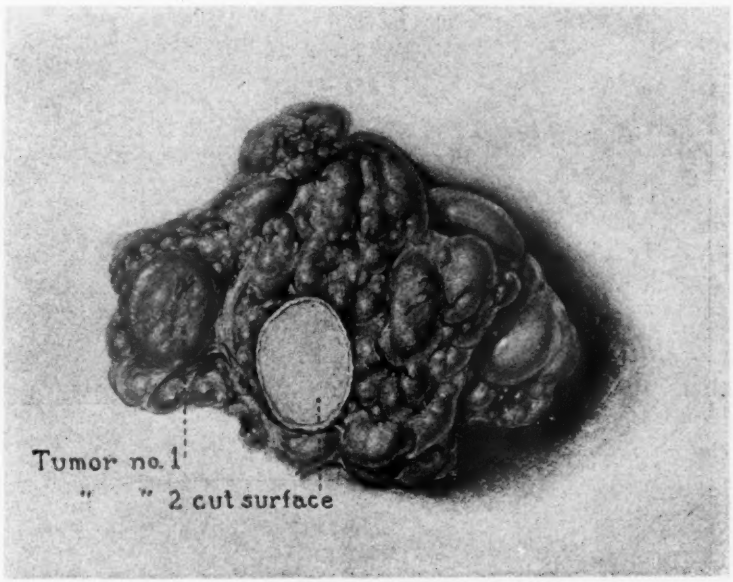

Fig. 10. Fibrochondroma of subpatella fat pad—multiple.

changes and swelling occur. This increase in size in an already crowded space causes pinching and irritation and the mass falls within a vicious cycle—its increase in size results in more compression and pinching and this in turn produces an increase in size.

Symptoms. Local sensitiveness on direct pressure; pain on full extension of the knee; pain and discomfort on ascending or descending stairs and grades, all of which are not constant in each case. The symptoms may be acute or chronic in type, and may present a varying and confusing picture as small pedunculated areas may become caught between the crucial ligaments and cause locking of the knee, or at least prevent the knee from being extended.

Treatment. In the acute cases rest is indicated with the knee slightly flexed, which position lessens the compression. After the acute swelling has subsided elevation of the heel to prevent full extension, as suggested by Sir Robert Jones, is an effective method of protection in convalescence. In a large percentage of cases a cure is the result.

Constant trauma from an occupation that requires a person to kneel often produces a hypertrophy of the pad and operative removal is required. A moderate number of the acute traumatic type do not undergo resolution and have to be treated operatively. Cystic and lipomatous tumors and calcified nodules occur, as illustrated; also, on two occasions I have operated on congenital anomalies in which small fibrous bands were attached to the lateral border of the subpatella pad and to the synovial tissue above the margin of the cartilage on the lateral surface of the condyle of the femur. These bands produced a catching of the knee by slipping over the condyle of the femur, producing an effect strikingly similar to that of a semilunar cartilage injury. There are many real pathologic lesions of the subpatella fat pad—traumatic, congenital and otherwise—which cause confusing but definite symptoms and disability. The removal of pathologic pads gives splendid results, but the excision of a normal fat pad as an excuse for doing something will not cure other lesions within the knee joint.

In my opinion there is a group of rare but existing traumatic synovial lesions which are not often recognized, namely, those of the so-called quadriceps pouch. Trauma to the quadriceps tendon and the synovial tissue beneath may tear these tissues, leaving one extremity attached and these tags may become hypertrophied and produce bizarre symptoms. On several occasions I have explored a knee because of a catching sensation about the patella and quadriceps tendons to find a synovial strip attached at its ends, hanging like a garland and catching under the patella. The operative removal of these is very gratifying to both the patient and the surgeon.

STABILIZATION OF THE ANKLE JOINT FOLLOWING MALUNITED FRACTURES

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PERHAPS there is no fracture which seems to be of such relatively little importance but which may prove to be so disabling as a fracture of the ankle. Fracture of the ankle, though a term frequently used, is really a misnomer. We are prone to use this terminology and not specify the exact type of fracture which an individual might have.

To treat properly fractures about the ankle, the physician in charge must have a complete knowledge of the anatomy about the joint, but since anyone interested in treating the ankle joint should know the anatomy we shall not deal with it here. Neither shall we deal with the various lines of forces producing fractures about the ankle joint. These forces have been dealt with extensively in the literature. A broken ankle must be efficiently treated, regardless of how it was produced. For the patient to have a painless, weight-bearing articulation, the various fractured surfaces about the joint must be replaced in their normal position. The alignment of the bone in the leg should be as nearly perfect as possible. Uneven joint surfaces and widening of the ankle mortice should be corrected, because these are the factors which produce discomfort after the healing period is over and convalescence completed.

CLASSIFICATION OF ANKLE JOINT FRACTURES

To approach the subject of stabilization of the ankle joint it is necessary to follow some definite classification, and in this manner review the various types of fractures and mention those particular fractures which may lead to a stabilization operation. A very satisfactory classification is one suggested by Dr. Frank D. Dickson.[1]

A. Fracture of the malleoli
   1. Isolated.
      a. Fibula.
      b. Internal malleolus.
   2. Combined.
      a. Lower bimalleolar without displacement of the astragalus.
      b. Bimalleolar with displacement of the astragalus.

B. Fracture of the weight-bearing surfaces of the tibia.
   1. Isolated.
      a. Posterior marginal fractures.
      b. Anterior marginal fractures.
   2. Combined.
      a. Anterior or posterior marginal fractures associated with fracture of the malleoli.

C. Tibiofibular separation.

FRACTURE OF FIBULA

Fracture of the lower end of the fibula does not often produce extensive disability unless it involves the extreme lower end of the bone, and incidentally produces a widening of the ankle mortice. This type of fracture occasionally goes unrecognized because of lack of *x*-rays taken in the proper positions. When displaced the fragment can be easily reduced in many instances into its normal position by a cast which extends from the knee down to and including the foot, with the foot in the inversion position. Untreated cases may require osteotomy of the lower end of the fibula to correct a valgus position of the foot and to correct widening of the ankle mortice. If this type of fracture is

properly treated early or if proper surgery is done, no disability should result. This type of fracture, in the experience of the writer, has never required stabilization of the ankle joint.

FRACTURE OF INTERNAL MALLEOLUS

Fracture of the internal malleolus carries with it a larger percentage of disability than does a fracture of the external malleolus. This is particularly true when the internal malleolus is fractured and involves with it a portion of the articular surface of the lower end of the tibia. Simple fractures of the internal malleolus are easily replaced into their normal position by applying a short leg plaster of Paris cast with the foot in extreme inversion and padded well with sheet wadding. However, when the internal malleolus is fractured and involves with it a portion of the articular surface of the tibia, other than that of the internal malleolus, not infrequently we find that it is impossible to replace it properly in its normal position.

When such complications ensue, a small incision over the internal malleolus should be made, and the fragment replaced and maintained in position by means of a small nail or some other means of internal fixation. Fractures about the internal malleolus when properly treated should not be followed by any disability. Untreated fractures of the internal malleolus, however, sometimes result in non-union and, consequently, misalignment of the foot and leg as a whole, with widening of the ankle mortice and a valgus position of the foot. This is more likely to be true if the individual has an already existing tendency toward valgus position of the foot. Open reduction in these cases is indicated. If the operation is successful there should be no disability later and, accordingly, no indication for stabilization of the ankle.

BIMALLEOLAR FRACTURE

Low bimalleolar fractures without displacement of the astragalus may be complicated if the two malleoli are fractured in such a manner that after one is properly replaced the other will not remain in its normal position. It may then be necessary to use some type of internal fixation for the internal malleolus and reduce the fracture of the external malleolus by the closed method. Frequently in this type of fracture there is also a separation of the lower end of the tibia and the fibula. When such is the case we should pad well the lower ends of these bones and, during the time that the plaster of Paris cast is hardening, force their ends together in an effort to narrow the ankle mortice if this has been separated at the time of the injury. Untreated fractures of this type usually require surgical intervention, but even then one can expect some remaining disability. However, it is unusual for this type of fracture ever to warrant a fusion operation of the joint. Immobilization above the knee joint is required, preferably with the knee flexed to prevent rotation of the foot outwardly.

Bimalleolar fractures with displacement of the astragalus are more difficult to treat. The treatment should be directed at replacing the malleoli in their normal position. If this cannot be done the physician should not hesitate to do an open reduction if he has the equipment and the experience. If the malleoli are properly approximated in their normal position it is not difficult to replace the astragalus. No disability should result after this type of fracture if the joint surfaces are properly replaced and good alignment of the leg and foot is achieved. The immobilization following such treatment should extend from above the knee with the knee flexed. Untreated cases of this kind frequently set up extensive disability. In many instances it is impossible to replace the malleoli and to overcome the displacement of the astragalus. If this type of fracture has existed for a year and if the patient is complaining of pain sufficient to warrant a stabilization of the ankle joint, the physician should do the operation.

FRACTURE OF TIBIA

Fracture of the weight-bearing surfaces of the tibia is one of the most important and one of the most disabling types of fractures about the ankle joint. Posterior marginal fractures may result in serious disability. The disability and the importance of this type of fracture depend on the amount of displacement of the posterior surface. If the posterior marginal fracture is small, manipulation may give satisfactory results. However, if the posterior marginal fracture is large, and the astragalus tends to slip into the line of fracture, it would seem that an open reduction is indicated. The posterior approach is the one of choice.

Unreduced fractures which have existed no longer than three months can probably be replaced surgically, and when properly treated carry very little, if any, disability. After the lapse of six to eight months, however, we do not believe that we are warranted in attempting to replace the posterior articular surface of the tibia. If the piece fractured is large and the astragalus is displaced, a stabilization of the ankle joint may be indicated, depending on clinical findings and the complaints of the patient.

Anterior marginal fractures are not frequent. If they cannot be reduced by the closed method, surgical intervention is indicated. The amount of disability remaining depends on the approximation of the fracture.

Anterior and posterior marginal fractures associated with fractures of the malleoli are difficult injuries to treat efficiently, particularly if the various pieces of bone are widely separated. In many instances it is absolutely impossible to replace these various fractured surfaces by the closed method and open operation becomes necessary. Just which fractures should be handled by the open method depends on the amount of displacement of the various pieces present about the joint. It would seem necessary to do an open reduction on the fragments not reducible by the closed method. Regardless of the method used, immobilization should be complete and should extend from the foot to above the knee, with the knee flexed. Speed states that 75 per cent of Cotton type fractures show poor results,[2] which would seem to indicate the advisability of radical treatment. When these fractures are seen early and several attempts to accomplish closed reduction have failed, one should not hesitate to use internal fixation of the posterior and the internal malleolus. The external malleolus can usually be sufficiently replaced to give a painless, functioning foot. Untreated cases of these fractures carry extensive disability and may require stabilization of the ankle joint.

TIBIOFIBULAR SEPARATION

Tibiofibular separation is frequently termed a "sprain of the ankle," because the condition is not recognized and the patient goes untreated. The separation is brought about by a rupture of the anterior and posterior ligaments of the external malleolus. Widening of the ankle mortice may produce a lasting disability, and such an injury should therefore be treated immediately by immobilizing the joint in a circular plaster of Paris cast with adequate sheet wadding and felt about the ankle. As the plaster of Paris cast is applied, the lower ends of the tibia and the fibula should be forced together and held there until a thick cast has completely hardened. When these cases are treated early, little if any disability should result. Neglected cases, in the experience of the writer, have never warranted surgical intervention. A Thomas heel, elevated on the inner border, with proper supports, often gives relief.

ADDITIONAL TYPE OF FRACTURE

The writer suggests that another heading be placed in Dickson's classification which would be entitled "Extensively

comminuted fractures of the lower end of the tibia, with fracture of the external malleolus." This is the most complicated type of injury which is found about the ankle, followed, in the author's experience, by much disability and many poor results. One may feel at a loss when first examining the *x*-rays of this type of fracture. However, an attempt at closed reduction should be made, as one is occasionally very pleasantly surprised to see how adequately all the fragments fall into their normal position. Closed reduction consists of more or less moulding of the fragments together. This is correct if the attempt is made immediately after the patient has been injured. These ankle fractures are usually placed in a position of slight inversion, the cast extending above the knee with the knee flexed. In this type of fracture stabilization of the ankle is frequently required, even if expert operators had been in charge. Neglected injuries of this nature almost invariably lead to stabilization, because of misalignment, widening of the ankle joint, and loss of smooth and even articular surfaces. No other type of fracture about the ankle so needs a stabilization operation.

To attempt to reduce this type of fracture weeks after the patient has sustained his injury is useless. Surgery must be done, but stabilization of the ankle joint is seldom indicated immediately after the injury has occurred. We feel that this is true because in many instances a patient has a good, functioning, painless ankle while the *x*-ray shows distortion and clinical examination shows a deformity about the joint. The writer feels that any fracture about the ankle joint possessing reasonable alignment and approximation should have a prolonged test at functional use before stabilization is advised. If the patient has attempted to use the ankle six months after convalescence is complete, and the ankle remains painful, while *x*-rays and clinical examination give reason to believe that the ankle is painful, stabilization is indicated. Those patients who have a gross deformity need not wait longer than three months after injury to undergo surgery.

The operation consists of completely denuding the articular surfaces of the fibula, the tibia and the astragalus. The literature indicates that either an anterior or a posterior approach may be used, but in the experience of the writer these incisions are not satisfactory because too much time is lost in waiting for a complete ankylosis after surgery. It is reasonable to believe that the deltoid ligament and the ligaments of the external malleolus would prevent thorough approximation of the denuded surfaces. It is also quite difficult to remove all the articulating surfaces on the posterior surface of the astragalus via an anterior approach; furthermore, it is quite difficult to remove the articulating surfaces of the anterior portion of the astragalus with a posterior approach without removing an unnecessary amount of bone. Consequently, a lateral approach has been used to the ankle joint.

An incision is made beginning from a point over the tarsal fossa extending backward and upward posterior to the external malleolus. The sheaths of the perineal tendons are not interfered with. The incision is made by sharp dissection, and courses very close to the lower end of the fibula. When the ankle joint is entered the foot is inverted. This gives a clear view of all the articulating surfaces making up the ankle joint, and they are easily removed in a very few minutes. The astragalus is replaced in the desired position, and the remaining raw surfaces of bone are easily held together. A long leg cast is then applied, with the foot plantar-flexed about 115 degrees, which allows for the heel of the shoe.

The patient is permitted to convalesce twelve to sixteen weeks, after which, if ankylosis is complete, a shoe is applied. The patient then continues on crutches until such time as he is able to discard them.

The stabilization operation seems, to those unfamiliar with it, to cause severe and extensive disability following this operation, but actually, on the contrary, many patients later walk with comparatively little limp. The mid-tarsal joints distal to the ankle loosen in a very satisfactory manner. If ankylosis is complete and the foot is placed in a true functioning position, these individuals are able to carry out almost any duties which they desire to attempt. The operation is advised only when there is extensive disability in the ankle and in those cases which have not responded to any conservative treatment. The operation is advised to overcome pain, weakness and insecurity.

If a patient who has had the stabilization operation of the ankle joint properly performed continues to have a permanent partial disability of 61 per cent as compared to an amputation at the ankle[3] then it would seem that there is no real indication for the operation. However, a person who is able to walk on his own foot without pain, without weakness, and with a negligible limp, apparently has a good foot with approximately 30 per cent disability as compared to an amputation at the ankle. If stabilization of the ankle joint will reduce an existing disability at the ankle from 75 to 85 per cent to a disability of 30 to 35 per cent, we feel that the operation is indicated and should be carried out.

COMMENT

The need for stabilization of the ankle joint would be lessened if those who treat fractures of the ankle would bear some of the following DON'TS in mind:

1. Don't wait for the swelling to go down before reducing fractures of the ankle.
2. Don't fail to *x*-ray the fractured ankle before and after setting.
3. Don't try to set a fracture of the ankle without an anesthetic.
4. Don't hestitate to attempt reduction more than once, but make these attempts early.
5. Don't use splints to maintain reduction. Use well padded circular casts.
6. Don't use short-leg casts except in simple fractures of the ankle. Apply the cast above the knee in extensive fractures about the ankle.
7. Don't fail to *x*-ray on the tenth day after reduction has been thought to be satisfactory.
8. Don't disregard the patient's complaint of pain about the posterior surface of the heel. A sloughing on the heel is disconcerting on "Settlement Day."
9. Don't hesitate to call a consultant or to refer a patient for additional care.
10. Don't fail to ascertain early if closed reduction is impossible. Operate within the first week if there are no contraindications.
11. Don't transport for long distances patients who have compound fractures without thoroughly cleansing the wound, preferably with tincture of green soap and water. Reduce protruding bones in the soft parts.
12. Don't neglect to pad well and completely immobilize the patient if he is to be transported.

SUMMARY

No injury about the ankle joint should be taken lightly. Injuries which may appear simple frequently carry a disability after the patient has attained his maximum recovery. A physician treating fractures of the ankle must have a sense of mechanics, and he can only understand the mechanics about the ankle by being thoroughly familiar with the anatomy. All fractures about the ankle joint should be reduced as soon as possible. One should not hesitate to try closed methods repeatedly; and, by the same reasoning, one should not hesitate to do an open reduction on fractures about the ankle if closed reduction is impossible. Comfortable, complete immobilization is of utmost importance in maintaining either closed or open reductions of fractures of the ankle. We suggest that a new heading

be included in Dr. Frank Dickson's classification of ankle joint fractures.

We believe that a lateral approach is preferable to either the anterior or the posterior approach. The lateral approach gives an ideal exposure of all the articular surfaces present in the joint. Stabilization of the ankle joint does not carry with it as extensive a disability as it would seem. Stabilization of the ankle joint should be advised only after several months of convalescence, and then should be carried out as a last resort.

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In fractures of the neck of the femur every movement of the body causes pain, so much so that it has been impossible to raise the patient for the use of the bed pan, and soiling of the bed has occurred

FRACTURE OF THE OS CALCIS

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THE subject of fracture of the os calcis has been of personal interest for a good many years. Whether it be a fresh fracture or an old fracture with pain and disability, the selection of the method of individual treatment is important. The mechanics of production of the fracture are practically always the same: a fall from a height, the body weight striking on the heel.

If one looks at the anatomy of the foot, one sees that the os calcis normally lies with about two-thirds of its entire bulk lateral to the mid weight-bearing line of the foot, and the other one-third medial to this line. The impact of the weight of the body against the heel drives the posterior two-thirds of the os calcis upward and outward and compresses it. The bone is of cancellous nature and practically never fractures twice in the same lines. The lines of fracture are irregular and are not in planes easily shown by an anteroposterior and lateral view in the *x*-ray. The impaction shortens the heel from front to back and widens it from side to side, and carries it upward and outward.

In view of the fact that the os calcis is shortened and the posterior two-thirds are driven upward toward the fibula, the foot is thrown into a pronated position, the longitudinal arch is lowered in proportion to the amount of upward displacement of the posterior fragments; the peroneal tendons are displaced outward or pinched between the lateral surface of the os calcis and the fibula. Further, whenever a fracture of the os calcis occurs there is immediate and rapid swelling around the posterior surface of the foot and ankle, which extends forward and upward, followed quickly by bleb formation if not checked.

There have been many methods devised to reduce this fracture and maintain it in reduction. This, on the face of it, implies that none of these methods is easy to apply with the assurance of a satisfactory result. What holds true with other conditions in medicine applies here—no two cases are exactly alike. Each must be analyzed and the necessities of each case must be considered. Methods mean nothing unless they are applied intelligently and are adapted to the case in hand.

Numerous difficulties are encountered in attempting reduction. Fracture of the os calcis is always an impacted fracture; further, it is impacted so firmly that great force properly applied is necessary to disimpact it, and without disimpaction nothing can be gained by any treatment. Tongs, mallets, wires or plaster casings are useless unless they tend to improve the position of the fragments and maintain them in the improved position.

From personal experience it seems that the first step in treatment is to prevent swelling and rapid bleb formation which generally occurs as an immediate result of this fracture. The most satisfactory method in our hands for meeting this requirement is a pillow splint as advocated by Gurd. When properly applied, this will absolutely prevent the occurrence of bleb formation and will reduce the swelling to a point where, within the short space of forty-eight hours, the treatment necessary for reduction of the fracture can be applied. Rather than permit swelling to occur, it is preferable to postpone the *x*-ray and put on a pillow splint or massive compression dressing promptly. The fragments do not change position; it is the swelling and bleb formation only that prevent reduction within a couple of days

after occurrence of the fracture. Should there be bleb formation, which cannot be cleared up in less than ten days or two weeks, treatment must be postponed until fixation can be applied. Therefore, if swelling is permitted to occur, reduction of the fracture must be deferred until such time as the skin is in condition to tolerate the application of some fixation.

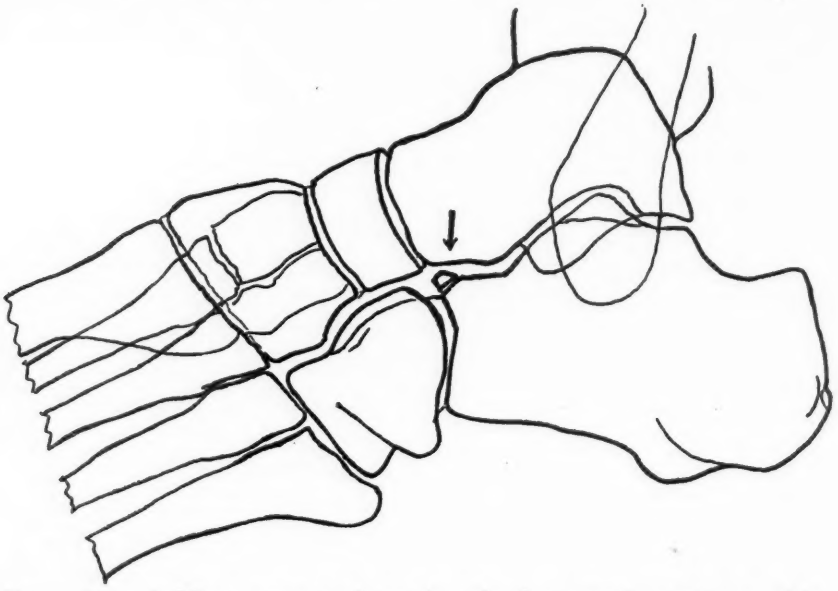

Fig. 1. Lateral-oblique *x*-ray to show triangular fracture of anterior os calcis.

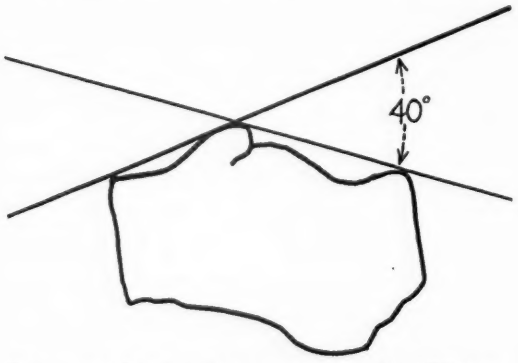

Fig. 2. "Salient angle" as described by Böhler.

Visualization by *x*-ray should always include stereolateral, lateral-oblique and anteroposterior views. A satisfactory anteroposterior film can be obtained by placing the plate against the sole of the foot, the patient in prone position, bringing the tube close to the back of the knee and projecting the ray downward parallel to the long axis of the tibia. This will give a view of the posterior two-thirds or three-fourths of the os calcis with the malleoli. The lateral-oblique (Fig. 1) is taken to show a small triangular piece which extends into the joint in the upper lateral surface. No other view will show this small fragment which, if it is present, forms a troublesome complication and will produce a permanent painful posterior tarsus. The perpendicular view will give information regarding the widening of the os calcis and the amount of displacement of the posterior two-thirds. The lateral stereo will show the lines of fracture into the astragalocalcaneal joint, as well as the amount of change in the salient angle, as described by Böhler. (Fig. 2.)

Böhler says, that "if two lines are drawn along the upper aspect of the calcaneus, one from the highest point to the anterior angle, and the other from the highest point to the upper part of the tuberosity, these two lines will normally make an angle of 140 to 160 degrees with each other, the complementary angle being 20 to 40 degrees. This angle, which is easily measured by the eye, is known as the tuberosity joint angle, or salient angle. In a fracture of the os calcis, the angle becomes smaller,

disappears, or is sometimes reversed. It is an important determination in recognition of fracture of the os calcis."

It must be remembered in all these views that the planes of fracture are not well defined, as in cortical bone, and the exits of lines of fracture on the various surfaces must be carefully studied to determine the number of fragments and the amount of compression, as well as the amount of displacement.

The same thing happens here that happens in Colles' fracture or any other compression of cancellous bone. The violence which causes the fracture and impacts the fragments actually disintegrates the bone cells, causing permanent loss of total length, so that no amount of manipulation will completely restore the bone to its normal shape. Therefore, we seek to accomplish certain things in reduction, namely:

(1) To break up the impaction, (2) to enable remoulding of the fragments downward and inward away from the external malleolus, and to restore them to their normal position in relation to the mid weight-bearing line of the foot; (3) to increase the height of the arch and to reëstablish the mechanics of the foot, enabling it to bear weight again without pinching the peroneal tendons, and to avoid throwing undue strain on any of the mechanical structures that support the weight of the body as it applies to the foot; (4) to restore the plantar fasciae to normal length.

Another complicating factor in this fracture is entrance of the fracture lines into the astragalocalcaneal joint. The fact that this so often occurs would seem to be one of the governing factors in the choice of method of treatment. An irregular, rough astragalocalcaneal joint with adhesions will probably produce pain regardless of the position of the os calcis. There is no doubt but that adhesions in this joint occur almost inevitably in fracture of this bone. In addition, there is thickening and induration which results in fibrosis of the internal and external lateral ligamentous supports, and these three things taken in combination always produce some limitation of lateral motion. Constant weight bearing subsequent to fracture, on a joint which is rough and irregular, will produce traumatic arthritis regardless of other conditions.

Treatment should be planned with reference to the amount of damage that has occurred to the joint at the time of fracture. How best to meet the requirements of each case is a matter of individual opinion. There are certain fundamental principles involved. The technique necessary to care for this particular fracture will have to be devised. It may be that none of the well publicized methods will meet the requirements and a combination of several will be necessary. Whether the impaction is broken up by striking the outer side of the heel with a mallet; whether it can be manually accomplished; or whether a carpenter's clamp is required, will be determined by the result of the operator's experience in using one or more of these methods. The fact remains that the impaction must be broken up completely in order to mould the os calcis back into shape and into position.

How best to hold it in position must also be determined at the time of manipulation. Too much pressure cannot be applied to hold the fragments, because the skin of the heel and sole will not tolerate undue pressure over even a limited period. A slough may be caused by too much force or too much pressure over too long a period, and a slough on the heel is a major disability in itself.

METHODS

In 1917 one of the authors[1] advocated cutting the tendo Achilles to relieve the fragments of the displacing muscle action of the gastrocnemius and soleus. This was very effective so far as maintenance of position of the fragments was concerned. It was discontinued when it was found that the calf muscles were permanently weakened as a result of the procedure.

They never returned to their full strength and consequently there was a disability in the leg, although the results so far as the os calcis was concerned were better than those obtained by any other method.

Pins and calipers of various kinds have been advised for the maintenance of fragments in position. It is true that there is usually one major posterior superior fragment, but this may be only one of a dozen fragments which, after the impaction is finally broken up, do not offer the possibility of being moulded into the normal contour by traction on one fragment only. If the os calcis were broken through the body in a clean transverse line, a pin or wire through the os calcis with weight applied sufficient to balance the pull of the gastrocnemius and soleus, would answer the purpose, but fractures of this type are seldom seen. A comminuted fracture cannot be reduced and all the fragments maintained in reduction by putting traction on one fragment even though it is of larger size than the others.

The treatment, then, resolves itself down to disimpaction of all fragments with proper moulding of these fragments into the normal angle and the normal position of the os calcis. There have been various methods advanced, all of which have strong points in their favor, and none of which will answer the requirements in every case. It is not possible in a paper such as this to offer a review of all the methods offered during recent years for treatment of these cases. Most of them have been devised at a time when it was necessary to meet the requirements of an individual fracture, and may not be applicable to the case at hand. The surgeon must therefore determine the requirements of his particular case before deciding what method or combination of methods will best suit his purpose.

In reviewing the various methods and from personal observation, the following appear to be the most logical procedures; they are based on variations of the same sound principles:

Hermann's Method. This author's[2] desire to reduce this fracture and hold it in reduction was prompted by the observation that in a large number of cases, 10 per cent developed sepsis where Kirschner wire or a Steinmann pin were used. He concluded that traction through a traumatized area was surgically unsafe, with which view we fully agree. He therefore first places the lower leg, ankle and foot in a compression dressing, preferably a pillow splint, to prevent swelling and bleb formation. When swelling has subsided sufficiently, the patient is anesthetized and turned on the side opposite the injury. A sandbag is placed under the inner surface of the injured heel: a tightly rolled linen towel is placed over the lateral surface in an oblique position with its lower end just below the tip of the external malleolus. Solid heavy blows from a moderately heavy rubber or felt hammer are struck on the towel below and behind the external malleolus until the impaction is completely broken up and until a depression can be felt beneath the tip of the external malleolus to about the depth of the surgeon's thumb. The heel is then grasped between the operator's hands and moulded downward and inward, motion of the subastragalar joint being tested at the same time. Following this, ice-tongs are placed in the large posterior superior fragment. The upper portion of a sawed-off crutch is placed just anterior to the border of the os calcis on the sole of the foot: the opposite end of this crutch is placed against the operator's chest, and the heel is pulled downward with the ice-tongs and further moulded. Hermann advises traction on the ice-tongs downward and outward, slowly changing the direction to inward. The ice-tongs are removed and further moulding is accomplished by lateral compression with a redresseur. *X*-rays are then made to be sure that a satisfactory reduction has been obtained. Small rolls of felt, measuring 4 by 1¼ inches are placed at a slight oblique angle beneath each malleolus. These pads, when

in place, serve as pressure pads to prevent excessive bone formation. A plaster boot is then applied, extending from the knee to the toes. The foot is held in slight inversion and extreme plantar flexion during the time the plaster is hardening.

Hermann feels that the after-care of these cases is the most important step of the entire procedure. The plaster and submalleolar pads are replaced every two weeks, care being taken to keep the foot at a right angle when the second plaster is applied. Constant snug pressure must be maintained below the malleoli. Following removal of the plaster at the end of ten or twelve weeks, a special ambulatory os calcis brace is applied. Daily massage and active foot and ankle motion are started; supination, plank walking, hot soaks and radiant heat are instituted. Weight-bearing is permitted within two weeks after removal of the last plaster, the shoe being fitted with an inner sole and $\frac{1}{16}$ inch lift on the inner margin of the Thomas heel. The splint is gradually discarded about eighteen weeks after the initial reduction.

Hermann reports 152 cases treated by this method, in 73 per cent of which the results were good, in 14 per cent fair and in 13 per cent poor. He believes the good results are due chiefly to the after-treatment which tends to maintain continued pressure beneath the malleoli and prevents piling up of bone.

The rationale of this treatment seems excellent. We have visited Hermann's clinic and have seen some of his cases, and were impressed by the thoughtfulness and care shown in the reduction and after-treatment. The principles upon which the treatment is based show an understanding of the problems involved.

Conn's Method. Conn[3] reported a series of cases in which he used four different methods, but he does not say upon what points of variation in the fractures he based his choice of method: (1) subastragalar arthrodesis, with or without regrooving for the peroneal tendons; (2) combined forcible lateral compression of the os calcis with skeletal traction; (3) a two-stage operation of lateral compression and skeletal traction for five weeks, followed by a combined subastragalar, astragaloscaphoid, and calcaneocuboid fusion; (4) on old cases in which a painful subastragalar motion was present, a triple arthrodesis. He feels that the results obtained were by far the best when the third method was used. Briefly, his technique is as follows:

A heavy wood clevis is applied just below both malleoli which, when tightened, acts as a disimpactor, inasmuch as it breaks up the bone so that it may be moulded easily. Following this step, steel fixation is used in the form of a Steinmann pin through the lower tibia and the tuberosity of the os calcis. Reduction is maintained by traction in opposite directions on these pins by a steel arc running posteriorly from one pin to the other and incorporated in the plaster boot. This position is continued for five weeks.

The second stage, at the end of five weeks, consists of fusion of the subastragalar, astragaloscaphoid and calcaneocuboid joints. All excess bone under the lateral malleolus is removed (Scoop operation). Three weeks later the plaster is removed and the position of the foot is observed. If it is not satisfactory or if there has been any slipping, the position can be corrected at this time. New plaster is then applied. Eight weeks after the fusion operation, weight-bearing is started slowly and cautiously, care being taken to prevent any undue strain on the longitudinal arch.

Conn credits his success to the fact that the depressed sustentaculum tali, which is so often overlooked, is recognized and corrected. In Conn's hands, this method gave better than average results, but it certainly constitutes a major surgical procedure in two stages. Judging from our personal experience with these fractures, this would not be justified in the average case. We believe that thoughtful treatment along less radical lines will produce as good

results, with less hazard of serious consequences as a result of infection around the Steinmann pin or infection at the time of the second stage operation.

Goff's Method. This method[4] is designed to allow the patient to be ambulatory within forty-eight hours after reduction is effected. Goff uses a carpenter's clamp, as described by Yergason, for both anteroposterior and lateral compression or disimpaction of the fracture. The clamp is used because of the greater force obtainable, which permits pulling the heel into position at once. Goff feels that this obviates the need for continued traction. Plaster is applied from toes to mid-thigh, with the knee flexed 20 degrees. The Böhler walking iron is incorporated immediately and the patient is permitted to walk within forty-eight hours, the weight being borne on the iron. At the end of the second week the plaster is cut below the knee and after ten weeks it is removed; full weight-bearing is permitted at the end of eleven weeks.

This procedure takes into consideration the remoulding and reposition of the fractured fragments of the os calcis. A great deal of force is used with the clamp to mould the fragments, or practically pinch them into shape. The flexion of the knee, the cast extending above onto the thigh, indicate that the author wishes to release the pull of the tendo Achilles, which seems important. However, even with a walking iron, if there is a tight fit of the plaster around the os calcis, as there must be to hold the fragments in position, the bearing of weight on the walking iron will force the cast upward as the weight of the body comes down against it; consequently there must be pressure on the inferior surface of the os calcis and a tendency to force it up into its old position.

The advisability of allowing a patient with a fractured os calcis to be ambulatory within forty-eight hours is questionable. True, it saves confinement and time in the hospital, and the companies who frequently have to pay for such injuries appreciate this reduction in hospital days, but from a surgical standpoint the welfare of the patient and securing a useful, painless weight-bearing foot, is much more important than the saving of a few hospital days. Our feeling has been that this procedure was rather radical as a primary method and should be resorted to only when more conservative methods have failed.

Böhler's Method. Probably more has been written on the Böhler method[5] of reduction and treatment of the fractured os calcis than on any other procedure. There have been frequent modifications of his original technique, by other surgeons and by Professor Böhler himself. His present treatment is approximately as follows:

In almost every case the injured leg is elevated on a Braun frame without traction. Light massage is instituted to decrease the amount of swelling. Usually, no skeletal reduction is attempted for eight or nine days, at which time, with the patient under spinal anesthesia, a Kirschner wire is inserted through the os calcis and an immediate reduction on a Böhler frame is performed. The line of pull for reduction depends on the type of fracture, the traction usually being at a 45 degree angle to the tibia, downward. From 55 to 60 pounds of traction is necessary to accomplish reduction. A Böhler redresseur is used for lateral compression. He has discontinued the use of the Kirschner wire through the tibia for countertraction because of the danger of infection in the tibial region and about the ankle joint, which he states he has observed rather frequently. He depends on countertraction from the flexed knee on the Böhler frame at the time of the initial reduction.

When reduction is obtained, a nonpadded skin-tight plaster is applied, carefully moulded about the foot and ankle. The leg is again put on a Braun frame, and traction of 15 pounds is applied on the Kirschner wire. This remains constant for six weeks, at which time the wire

is removed and a new plaster is applied, into which is incorporated a walking iron. This is removed six weeks later. At the end of fourteen to sixteen weeks after the initial reduction, the plaster and walking iron are discarded and full weight-bearing is allowed. The patient is fitted with a shoe containing an inner sole to maintain the newly formed longitudinal arch.

Böhler's method has proved very satisfactory in the hands of a number of surgeons. One of the authors has observed Böhler's work and results within the last year. Böhler himself, however, gives no such rosy and ideal picture of the results of this method as seems to be prevalent in this country. This is probably due to the fact that the Böhler apparatus and method have been publicized to a large extent through the sale of the equipment which he has designed and used. The occurrence of traumatic arthritis is frequent. Böhler cautions against the overuse of the redresseur because of the possibility of slough. He also cautions against too early weight-bearing. He had not been using a compression dressing to prevent bleb formation, but elevated the foot for ten days before starting treatment. Extension was maintained and the patient was kept in bed for six weeks following reduction, after which a skin-tight plaster was applied. Note is made here that neither weight-bearing nor dependent position was allowed until all possibility of displacement of the fragments and undue swelling had been eliminated by time.

Wilson's Method. Wilson,[6] in 1927, concluded that it was almost impossible to get anatomic reduction in fracture of the os calcis, and advised immediate subastragalar arthrodesis. This method he followed for some time. He exhibited very satisfactory results so far as painlessness was concerned, but the deformity produced by impaction and displacement of the fragments was not changed, except insofar as the os calcis was moved somewhat inward at the time of operation, and the heel inverted somewhat more than is usually the case immediately following fracture. Wilson's results were probably due to the fact that at the time of operation he was able to restore, more or less, the weight-bearing position of the os calcis, with its two-thirds lateral to the mid weight-bearing line and one-third medial thereto; also the whole bone was moved medially at the time of operation.

This is a valuable operation and is necessary in probably 30 to 35 per cent of cases even after proper reduction, but in our opinion it should be postponed until the fracture is reduced and healed in as near anatomic position as it is possible to attain. Then, if the patient has a painful astragalocalcaneal joint, Wilson's procedure should be followed. In these cases, the operation properly performed gives a highly satisfactory result after ankylosis of the joint occurs.

In 1923, one of the authors[7] advocated an operation for the relief of disability in old fractures of the os calcis, which attempted to do what Wilson later effected with an arthrodesis. In this operation the mass of callus which lies behind and beneath the external malleolus, which is an outgrowth of the widening and fracture of the os calcis, was removed with a gouge, and the groove left much deeper than normal. This part of the operation has been done frequently, and is advocated by Cotton and others. This, however, was considered by us only the first step in the operation. The second step has apparently never been given consideration either in the literature or in discussion, and the second step is just as important as the first, if not more so.

After the mass of bone had been removed from behind and beneath the external malleolus and the lateral supports of the ankle severed in getting to this excess callus, a Thomas wrench was applied with the proximal arm lying along the outer margin of the foot over the os calcis, extending forward over the fifth metatarsal, the distal arm lying against the medial side of the astragalus just below

the internal malleolus. With strong, smooth, steady pressure the foot was inverted, and with it the os calcis was carried medially. Frequently in this procedure a ripping can be felt and heard as the adhesions between the os calcis and the astragalus are broken up. When the heel can be moved inward and maintained in that position by pressure on the lateral side of the heel, the wound is closed and the foot fixed with the heel in inversion. The height of the arch is increased by firm eversion of the part of the foot anterior to the astragalus.

It was found that a skin-tight plaster, applied to the lower leg and heel and held firmly until it was hard, was the easiest method of maintaining the heel in its position. The anterior part of the cast, with eversion of the front of the foot, was applied after the posterior half had hardened. In this way the height of the arch was increased, the os calcis was moved over to the midline, and pinching of the peroneal tendons was relieved.

The results of this treatment have proved highly satisfactory. The so-called regrooving operation is incomplete. The inversion of the heel should be performed at the same time.

SUMMARY

There has been a voluminous literature on fracture of the os calcis in the last ten years. The results of treatment of this injury have improved, but the results will never be perfect in the hands of anyone, or as a result of the use of any one method. It is a fracture in which a certain amount of bone has been destroyed. It is comminuted in cancellous bone, and the major fragment is attached to a tendon controlled by two of the strongest muscles of the body. The bone must bear weight and be subject to the trauma of weight-bearing. To restore painless function implies perfect mechanical restoration of bone, which frequently is impossible; it also implies a freedom of motion between the os calcis and the astragalus which is practically always limited, regardless of the methods used in reduction and maintenance of reduction. Therefore, ideal results are almost impossible to attain. The future will present better results, however, if careful analysis of each case is made, and if the choice of treatment or combination of treatment is suited to and suitable for the individual case.

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COMPRESSION SPRAIN OF THE FOOT

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DURING the past year, in an active industrial practice, a sufficient number of cases of a similar, but unusual type of injury to the foot have been seen to warrant description as an entity. The mechanism of injury in the first few cases suggested the term "hand truck injury" but subsequent cases showed that the injury could be produced in various ways, therefore leading to the title above.

The majority of these cases were caused by the small iron wheel of a hand truck striking the back of the foot. Sometimes, the four-wheel truck was pulled by the subject himself; in other cases the injured was overtaken by a two-wheel truck pushed by a fellow employee. To produce the typical injury, it is necessary that the fore portion of the foot be fixed, either being jammed against some immovable object or bearing the weight of the body on the ball of the foot, at the moment of impact. When the wheel strikes, it does not force the os calcis completely down, but rather propels the foot and leg forward over the fixed anterior portion of the foot, thus elevating the longitudinal arch rather than depressing it.

Clinically, there is immediate pain, swelling and ecchymosis, but little or no deformity. *X*-ray examination may reveal multiple fractures involving any of the bones. Most typical in the cases observed were chip fractures into one or more of the tarsometatarsal joint spaces. Several patients revealed fractures of the shafts of the metatarsal bones without displacement and one revealed a chip fracture of the head of the astragalus.

Of fourteen such injuries observed during 1937, three representative cases are summarized briefly in order to illustrate the mechanism of the injury and the clinical course:

CASE I. While the patient was pulling a hand truck loaded with sheet iron, one sheet fell to the platform in such a way that the toe of his left foot became jammed against the iron, following which a wheel of the truck compressed the foot from behind. An *x*-ray film revealed displaced chip fractures of the bases of the third and fourth metatarsal bones. The foot was immobilized in a plaster of Paris cast for one month and the patient returned to work after eight and one-half weeks.

X-ray examination four months following injury revealed the fractures to be well healed, but disclosed extreme osteoporosis of all the tarsal bone, but not of the metatarsal bones. Clinically, the disability was out of all proportion to the severity of the fractures. The patient was finally discharged with an estimated disability of 20 per cent loss of use of the foot.

CASE II. This patient had a head-on collision while driving an automobile. At the moment of impact his right foot was pressing on the brake pedal just forward of the heel of his shoe. The impact forced the toe of his shoe against the dashboard, thus causing the weight of the body to be placed on the right foot, with the pedal under the arch as a fulcrum. The arch of the foot was elevated and the foot compressed in its long diameter. *X*-ray examination revealed typical chip fractures of the bases of the first four metatarsal bones. The foot was immobilized and temporary disability of eight and one-half weeks was suffered. Following this, there were complaints of persistent pain and swelling.

X-ray examination three months after injury revealed osteoporosis of the metatarsal and tarsal bones. The fractures healed in good position. Permanent disability was estimated at 20 per cent loss of use of the foot.

CASE III. The patient was assisting in pushing a motor truck. As he braced himself on the ball of his right foot, the left rear wheel

equipped with a traction-grip tread, caught the back of his foot and thus squeezed it downward and forward toward the fixed anterior portion of the foot. This patient was wearing a heavy work shoe with a built-in steel arch at the time of the injury. The nature of the injury is shown by the fact that after his shoe was removed, fellow workmen had to remove this arch with a sledge hammer so that the subject could wear the shoe while he was being transported to the doctor's office. Immediate *x*-ray films were made and revealed transverse fractures of the base of the fifth and of the distal third of the second metatarsal bones. There was no displacement of the fractured segments.

The foot was immobilized in plaster of Paris for four weeks. This was followed by baking, massage and the application of a moulded felt pad beneath the arch. He returned to work after eight weeks. Induration and stiffness of the foot persisted. Ankle motion was preserved, but motion of the subastragalar and midtarsal joints was practically nil.

X-ray films made two months after injury revealed extreme osteoporosis of the tarsal bones with narrowing of all joint spaces, particularly the subastragalar. The fractures had healed perfectly without displacement. *X*-ray examination four months after the injury revealed the bones of the foot to be practically normal in appearance. Clinically, the subastragalar motion was limited 50 per cent and the patient complained of considerable pain. He was discharged with an estimate of 30 per cent permanent loss of use of the foot.

SUMMARY

"Compression sprain" of the foot is described as a typical injury, as it is initiated by compression of the foot in its long axis, elevating the longitudinal arch sufficiently to cause a severe sprain of all of the joints in the middle of the foot with chip fractures into these joints. This is particularly true regarding the bases of the metatarsal bones. The disability in such cases, both in length and severity, has been far out of proportion to the fractures suffered. Heretofore, too much emphasis has been placed on the fractures, but in the future, these cases will be treated primarily as those of sprain. Active motion and vigorous physical therapy should be instituted with immobilization of the length of time for that of sprains. Success in treatment depends on limiting the period of complete immobilization.

EMBRYOLOGIC AND ANATOMIC CONSIDERATIONS IN ETIOLOGY OF INGUINAL AND FEMORAL HERNIAS

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CONTRARY to general opinion, nothing is more erroneous than the belief among some physicians and the laity that muscular effort is the cause of indirect inguinal hernia. The popular idea of the modus operandi is that a sudden or long-continued muscular effort, in some unexplainable manner, causes a pressure on the abdominal contents and this in turn forces a knuckle or loop of intestine to make a passage through the solid abdominal wall, regardless of the intricate network of muscles and fascia effectively arranged to prevent just such an accident. How anyone can seriously believe that a soft, velvety, collapsed coil of intestine can suddenly assume the hardness of a cold chisel and accomplish something the human finger cannot do, is beyond my comprehension. It is a well known fact that the finger can not be forced through the internal inguinal ring if there is no empty sac opening into the abdominal cavity.

In postoperative ventral hernia, a large, fully developed sac never appears suddenly—the peritoneum must be subjected to weeks or months of continuous stretching before a sac is formed—and yet the muscular protection over the inguinal canal is many times stronger than that over a thin abdominal scar.

If simple increase in intra-abdominal pressure could cause a hernia we would all have them, and they certainly would appear at the weakest points in the abdominal wall and not at the strongest—the inguinal opening. The opening in the diaphragm for the esophagus is an ideal weak point where hernias should frequently appear—but they do not. The femoral ring is the weakest spot in the lower abdominal wall—a large ring with rigid walls and no muscles to guard it—yet hernia here is rare in men.

In spite of the popular notion, the inguinal canal is not weak. It is so powerfully constructed that if there is no patent processus vaginalis waiting for a tenant, it is impossible to produce an indirect inguinal hernia no matter how much force is exerted by lifting, straining, or any other form of violent effort. The semi-sphincteric fibers of the internal oblique exert a lifting power, which is called into play during increased intra-abdominal pressure, and this brings into action a corresponding amount of resistance over the inguinal canal.

Once the internal ring relaxes or enlarges, and a tongue or wedge-shaped piece of omentum gains an entrance, it is an easy matter for the mass to dilate the internal ring to a point where it has no ability to come back or regain its tone, and the protection of the semi-sphincter action of the internal oblique and transversalis muscles and Poupart's ligament is lost. Once past the internal ring, the hernia rapidly dilates the inguinal canal on its way to the external ring.

In 1817, Cloquet, a French anatomist, in the course of 500 dissections, found a depression of the lower border of the internal ring with more or less bulging of the peritoneum. He called this "the infundibuliform process," and found it so often that he thought it a normal condition! We now know that he found an open processus vaginalis. The obliteration of the processus vaginalis is best studied during hernia operations on infants and children. When the processus vaginalis is obliterated only in its lower half or two-thirds, it is easy to find a well-marked fibrous cord springing from the fundus

of the sac at its lowest point. This cord can be traced down in the spermatic structures to the tunica vaginalis testis. Sometimes this obliterated portion of the processus vaginalis resembles the tail of a kite; often a well-defined mass of scar tissue is the dividing line between the open and the obliterated portion of the processus vaginalis.

The congenital origin of indirect inguinal hernia is shown by the uniform arrangement of the vas and blood vessels in relation to the sac—the vas is always to the inner side and firmly adherent to the sac regardless of how recent is the discovery of the hernia. The vessels are always behind the sac and separated by an interval from the vas. Small sacs lie right in the heart of the spermatic cord—a strange location for an "acquired" hernial sac. At the same time the highly organized tissues and delicate blood vessels over the sac and in the cord show no evidence of a sudden descent of a hernial sac. Nothing but a preformed sac can explain a hernia that protrudes to the external ring on its first appearance. Peritoneum requires weeks or months for stretching.

EMBRYOLOGY

Descent of the Testicle. Little has been added to our knowledge of the descent of the testicle since the observations published by John Hunter in 1762. In early fetal life the testes are located at the back part of the abdominal cavity, behind the peritoneum and below the kidneys, about on a level with the upper lumbar vertebrae. The anterior surface and sides are covered with peritoneum. By the second fetal month the testis and epididymis are attached to the genito-inguinal ligament. This ligament, which is at first a slender band, soon becomes well defined, and stretches from the testis to the lower part of the anterior abdominal wall where the scrotum is later formed. During the third fetal month the testis, guided by the ligament, descends to a position which corresponds to the internal abdominal ring.

With advancing growth, the peritoneum covering the testis envelops it entirely and forms a mesentery, and the muscular, fascial and peritoneal layers of the anterior abdominal wall show a protrusion which results in the production of a sac—the inguinal bursa. This deepens and extends into the scrotal fold, which meanwhile is formed independently as an integumentary fold. The genito-inguinal ligament, being attached to the structures undergoing invagination, extends into the inguinal bursa. The muscular tissue of the wall of the bursa is derived from the internal oblique and transversalis, and constitutes the cremaster. The lining of the inguinal bursa is the direct continuation of the general serous membrane of the abdominal cavity, and later it constitutes the processus vaginalis peritonei. By the fifth month the upper part of the genito-inguinal ligament has disappeared and the lower portion has become a thick cord and produces an elevation of the floor of the bursa—the inguinal conus. During the seventh and eighth months the inguinal conus and attached testis are drawn downward into the inguinal canal and through it, until shortly before birth the testis reaches its position in the scrotum.

The inguinal conus and the genito-inguinal ligament constitute the gubernaculum testis, which becomes shorter and smaller as the testis descends. The remains of the gubernaculum testis constitute the scrotal ligament, the subserous band which permanently attaches the tunica vaginalis and testicle to the surrounding tissue of the walls of the scrotum.

The retroperitoneal position of the testis is always retained, the testis and the accompanying constituents of the spermatic cord descending outside of the peritoneal pouch, which extends into the scrotum. For a time free intra-abdominal communication is maintained by the now tubular processus vaginalis; usually, however, by the time of birth, or shortly after, this canal is obliterated, the isolated,

lower end of the peritoneal process persisting as the pouch of the tunica vaginalis which almost surrounds the testis.

The Processus Vaginalis. The processus vaginalis is the entire process of peritoneum that accompanies the testis and cord. It is divided into two portions: the funicular process, which invests the cord, and the tunica vaginalis testis, which surrounds the testis.

The processus vaginalis or funicular process, which appears at the third month of intrauterine life, is a canal or peritoneal diverticulum that entirely surrounds the testis and its epididymis, and is continuous with the peritoneal cavity. The relation of the processus vaginalis to the testis has been a subject of considerable controversy: Kölliker and Henle believed that the processus vaginalis is formed before the descent of the testis and that it has no relation to the latter. This theory has been generally abandoned following the investigations of Bramann, Sachs, Russell, Murray and others, who demonstrated that the processus vaginalis does not exist before the descent of the testis, and that the gubernaculum testis is responsible for the migration of the testis and also for the descent of the processus vaginalis, which reaches the bottom of the scrotum in advance of the testis.

Whatever may be the mode of its formation, the funicular process tends to become obliterated after birth. Hunter believed the obliteration begins in the upper part of the vaginal process; Jarjavay thought it started in the middle part; Féré maintained that the lower portion is the first to close. In women, patency of the canal of Nuck is a cause of congenital hernia; however, this condition is rare, because of the narrowness of the canal and the small internal opening.

Patency of the Processus Vaginalis. Camper, in 1785, in a series of dissections in infants, found the processus vaginalis open on a single side in 31.5 per cent of the subjects, and open on both sides in 45 per cent. Keith stated that in children 3 to 4 months old, the processus vaginalis is open in 30 to 40 per cent of all cases. In infants under 4 months old, Sachs found obliteration in 41 per cent, and permeability in 30 per cent. After the fourth month the percentage of closures was higher, obliteration being found in 69 per cent, and permeability in only 4 per cent.

In 1899 Russell stated that all inguinal hernias in both adults and children were congenital in origin and were due to a persistent patent funicular process. All degrees of patency may be encountered.

The Obliteration of the Processus Vaginalis. The obliteration of the processus vaginalis occurs at two points, first at the internal abdominal ring, and a little later at a point just above the epididymis. Between these points, the processus vaginalis is an isolated tube which becomes smaller, its walls are occluded, and it finally terminates in a small fibrous cord, which is easily recognized during dissection or operation.

If the processus vaginalis does not become obliterated, a congenital hernia sac results, which may become a hernia, either at birth or in later life. During their descent the testis and spermatic cord obtain more or less extensive investments of such parts of the abdominal walls as have taken part in the formation of the original bursa inguinalis.

The Canal of Nuck. A peritoneal diverticulum in the inguinal canal in women, which was first described by Nuck in 1672, and corresponds to the processus vaginalis testes in man, is the cause of indirect inguinal hernia in females. The canal of Nuck is normally obliterated about the seventh month of intrauterine life, earlier than in the case with the processus vaginalis testis. The patency of this process was noted in 18 per cent of the subjects examined by Féré, in 24 per cent of those examined by Zuckerkandl, and in 25 per cent of those examined by Sachs.

The Gubernaculum in the Female. In the female, also, there is a gubernaculum that influences the position of the ovary to a

certain extent. The remains of the gubernaculum lying between the attachment of the cord to the uterus and its termination in the labium majus, form the round ligament of the uterus.

Indirect Inguinal Hernia in the Female. The inguinal canal in the female is longer and narrower than in the male. It gives passage to the round ligament and its vessels, to the genital branch of the genitocrural nerve, and sometimes to a peritoneal diverticulum (the canal of Nuck). After leaving the external ring the hernia passes into the upper part of the labium majus. If it remains incomplete, it is a bubonocele or pubic hernia, and its oval or globular shape resembles a direct inguinal hernia in the male. Incomplete hernia is the form commonly found in the female, and it does not become labial, as frequently as indirect inguinal hernia in the male becomes scrotal.

ANATOMIC CAUSES OF INDIRECT INGUINAL HERNIA

The anatomic factors that favor the development of indirect inguinal hernia may be summarized as follows: (a) the descent of the testis which carries with it a process of peritoneum, transversalis fascia (infundibuliform fascia), and cremaster muscle; (b) the weak spot at the internal ring, caused by the pouching of the peritoneum; (c) the location of the internal ring, which makes it unable to resist sudden increase in intra-abdominal pressure caused by straining, coughing, whooping cough, pregnancy, tight lacing, obesity, ascites and tumors; (d) the hernia cannot break through outside the internal ring because of the well-developed iliac and transversalis fascia; (e) the fact that there is usually a weak point near the inner side of the internal ring that is unprotected by muscle or tendon; (f) the existence of an unobliterated processus vaginalis or a preformed sac.

Deficiency of the Conjoined Tendon. Absence or weakness of the conjoined tendon may be an important factor in the causation of direct hernia, and of less importance in the etiology of indirect hernia.

Deficiency of the Internal Oblique and Transversalis Muscles. Ferguson observed that during operations for indirect inguinal hernia, there is frequently a deficiency of the internal oblique and transversalis muscles at their origina from Poupart's ligament, and he concluded that the faulty development of these muscles weakens the internal ring, and is the most common cause of indirect inguinal hernia.

Lipoma in the Inguinal Canal. A lipoma in the inguinal canal that is adherent to the sac or cord is almost always attached to the properitoneal fat at the internal ring, and by causing a bulging into the canal, favors the development of hernia. The lipomata should always be removed at operation to lessen the danger of recurrence. In 154 hernia operations, Speed found lipomata in 47.4 per cent.

The External, Superficial or Subcutaneous Abdominal Ring. The external ring or opening in the aponeurosis of the external oblique is located just above and to the outer side of the crest of the os pubis. This aperture is oblique in direction, triangular in shape, and usually measures about an inch from base to apex, and about half an inch from side to side. The normal external ring will seldom admit the tip of the little finger and it is impossible to palpate the inguinal canal or the internal ring. When the external ring is large the cremaster muscle is well developed, apparently an attempt of nature to make up for the weakness in the aponeurosis of the external oblique.

The Internal or Deep Abdominal Ring. The internal ring can be located on the external abdominal wall by a circle three-fourth of an inch in circumference, placed about three-fourth of an inch above the middle of Poupart's ligament.

The Inguinal Canal. The inguinal canal is an oblique opening about 1½ inches long, and is situated parallel to the inner half of Poupart's ligament and a little

above it. It is directed downward and inward and extends from the internal or deep abdominal ring to the external or superficial abdominal ring. It is 1½ inches long in the male, and 2 inches long in the female, the greater length in the female being due to the wider separation of the iliac bones. In large hernias the obliquity of the canal diminishes and the two rings almost meet.

In the male, after the descent of the testis at about the sixth month, the canal contains the ilio-inguinal nerve, the genital branch of the genitocrural nerve, and the spermatic cord; in the female, it contains the round ligament, the ilio-inguinal nerve, and the genital branch of the genitocrural nerve. The anterior and posterior walls of the canal run obliquely downward, inward and forward, and lie in apposition except for the space occupied by the cord or the round ligament.

The inguinal canal is bounded in front by the aponeurosis of the external oblique throughout its entire length, and by the lower fibers of the internal oblique at its outer third; behind, by the triangular ligament of the abdominal wall at its inner end, and by the conjoined tendon and transversalis fascia; above, by the arched fibers of the internal oblique and transversalis muscles; below by Poupart's ligament, as far as the posterior shelving margin which gives attachment to the transversalis fascia; and at its inner end by Gimbernat's ligament. This lower boundary is sometimes called the posterior wall of the inguinal canal.

Predisposing Causes. There are several factors that may favor the production of indirect and direct inguinal hernia:

1. Heredity. The influence of heredity as a cause of inguinal hernia is uncertain. Cooper believed that the shape of the pelvis plays some part. Macready said that hernia occurs more frequently in subjects whose parents had hernias then in those whose parents were sound. It is probable, however, that an inherited weakness of the abdominal muscles is just as much of an influence as the existence of hernia in one or both parents.

2. Sex. Inguinal hernia is very much more common in males than in females. Ninety-five per cent of all hernias in males are of the inguinal type, while in females only 45 per cent are inguinal. Macready, indeed, found that 97.5 per cent of all hernias in men, and 60.3 per cent of all hernias in women, are inguinal. The greater prevalence in the male is partly due to the size of the spermatic cord, which is much larger than the round ligament, and the processus vaginalis testis is less frequently obliterated than the canal of Nuck.

3. Age. Macready also stated that in the first year of life a larger number of persons are living; consequently there are more hernias then than in any subsequent year. In the first year, 17.5 per cent of males have hernia, and 9.16 per cent of females.

The number of hernias in children and adults is highest during the years of greatest activity. The tendency to hernia increases with age, but the exciting causes are less active. After fifty years, the reduction in the number of hernias in women is much greater than in men.

Frequency of Right and Left Hernias. The greater frequency of right hernia in the first year, and the high proportion until after puberty is due to delayed descent of the testis and to the later closure of the canal on the right side. After the age of 20, hernias occur on the left side nearly as frequently as on the right side.

Congenital Potential Hernia. The presence of empty congenital sacs in adults is more common than is generally supposed. Many people carry these potential hernial sacs throughout life without evidence of hernia.

In the post-mortem examination of 100 subjects who presented no evidence of hernia during life, Murray found sacs in twenty-one; in several of these there was more than one sac. In another series of 200 examinations, Raw and Murray

found sixty-eight peritoneal diverticula, and of these fifty-two were femoral, thirteen inguinal, and three umbilical. Murray believed that when a patent funicular process exists, the occurrence of a hernia depends on the size of the opening at the internal abdominal ring and the strength of the muscles that protect this orifice. He also stated that when the processus vaginalis has been completely obliterated, it is impossible for an indirect inguinal hernia to occur.

Hernias are most likely to come down into preformed sacs following the activities of youth, the laborious occupations of adult life, and the strain of repeated pregnancies. Emaciation, due to disease or old age, may also be a factor by removing the fatty plugs that have kept the patent funicular process temporarily closed. Owing to the lack of food during the World War, there was an increase of 25 per cent of hernia in Germany alone.

It is not unusual to see an indirect inguinal hernia develop on the sound side after one on the opposite side has been reduced and retained by a truss or cured by operation. This second hernia is probably due to an increase in intra-abdominal tension. I often advocate a bilateral operation or injection treatment for inguinal hernias, especially in young adults, since in operating for unilateral hernia, I have found a potential sac on the opposite side in 40 per cent of all cases. I believe it is always advisable to operate on the sound side when there is an enlarged ring, or an exaggerated impulse. This is demanded by some State Industrial Commissions.

Formation of Indirect Inguinal Hernia. It is generally believed that all indirect inguinal hernias are due to a preformed sac which consists of an unobliterated portion of the processus vaginalis. Given an exciting cause that increases intra-abdominal tension, the preformed sac together with a knuckle of small intestine or a piece of omentum is forced through the internal ring. The sac enters the inguinal canal above the cord, and descends in front of it. While a slight bulge over the internal ring may remain unchanged in size for years, once the sac has passed the ring it has overcome the point of greatest resistance, and its progress down the inguinal canal is rapid. If the pillars of the external ring are well developed and the opening only large enough to allow the passage of the cord, the hernia may be temporarily checked in its descent at this point. Beyond the external ring, the coverings of the sac exert only very little resistance and the hernia quickly descends to a position opposite the upper level of the testis, where the firm connections between the coverings of the cord and the tunica vaginalis may check its progress. If it overcomes the resistance here, it usually goes to the bottom of the scrotum and is known as a scrotal hernia.

In complete hernias, descent is probably aided by the weight of the hernial contents and by the mesentery which elongates and ceases to lend its support to the intestines. In females the hernia follows the round ligament through the inguinal canal into the labium majus.

The obliquity of the inguinal canal is a natural obstacle to hernia, because an increase in intra-abdominal tension forces the inguinal walls more firmly together. When a patient strains, the conjoined tendon and transversalis contract vigorously, and if a finger is inserted in the inguinal canal, it is firmly gripped between the conjoined tendon and Poupart's ligament. Keith stated that intra-abdominal pressure is raised to 100 mm. of mercury by straining or lifting. Congenital hernias (hernias into a performed sac) usually follow a sudden increase in intra-abdominal tension which is sufficient to overcome the resistance offered by the valve-like action of the inguinal canal.

In addition, there are often predisposing causes, such as a patent processus vaginalis, a depression in the infundibuliform fascia, a weak point in the abdominal wall, congenital or acquired weakness of the

abdominal muscles, emphysema, long-continued increased intra-abdominal tension, prostatic disease or urethral stricture. In the cadaver, with the inguinal canal opened, intestine cannot be drawn into the scrotum on account of the shortness of the mesentery.

Direct Inguinal Hernia. Direct inguinal hernia is so named because it takes the most direct route through the abdominal wall, passing through only the lower fifth of the inguinal canal.

The weak spot of the middle fossa is the usual point of exit of direct hernia. It is directly behind the lower fifth of the inguinal canal and the upper edge of the external ring between the outer edge of the rectus muscle and the deep epigastric artery, at a point where the abdominal wall contains no muscular layer, being weakened anteriorly by the gap in the external oblique aponeurosis at the external ring. The thinnest point, however, is at the upper and outer angle of the external ring where the posterior wall of the canal is not reënforced by the conjoined tendon or the triangular (Colles') ligament.

Direct hernias usually break through into the inguinal canal because the resistance is less in that direction than it is inward toward the rectus muscle. These hernias are never congenital and they have no preëxisting path. They are of slow development and ordinarily occur in adults. They are characterized by a small sac with a relatively large opening, and because of the shortness of the neck of the sac, they are globular in shape. Direct hernias generally remain bubonoceles, and on reduction go directly backward into the abdomen. The abdominal opening is easily felt, as it has the outer edge of the rectus muscle to the inner side and the crest of the pubis below. Direct hernias rarely descend into the scrotum.

Inguinal Hernia in the Female. Inguinal hernia constitutes about 45 per cent of all hernias occurring in females. Macready estimated the frequency at 60 per cent. These hernias are almost always of the indirect variety and are of congenital origin, being due to an unobliterated process of peritoneum, known as the canal of Nuck, which is analogous to the processus vaginalis testis. Obliteration of this peritoneal diverticulum occurs with greater frequency in the female than in the male. Nobbe stated that the canal of Nuck is found in 8 to 10 per cent of female subjects, and a patent processus vaginalis testis is present in 30 to 40 per cent of male subjects.

Direct Inguinal Hernia in Women. Direct inguinal hernia is less common in women than in men, because in women the shape and formation of the inguinal canal are different, the external ring is smaller, and the aponeurosis of the external oblique covering the inguinal canal is stronger.

Microscopic Examination of the Sac. Microscopic examination of a hernial sac shows that it consists of a thin lining of endothelium lying on a thick layer of dense fibrous tissue.

Hertzler remarks that one of the strongest evidences of the fact that the sac is preformed is afforded by an examination of the connective tissue at the point of union of the sac and cord. Microscopic observation will show an interlacement of fibrils running parallel to the walls of the sac and continuing over the cord, but separated entirely from other surrounding tissue. In several hernias of less than five days' duration, microscopic examination showed that the union of the sac to the cord was made up of fully developed fibrous tissue free from cellular infiltration.

Femoral Hernia. The congenital origin of femoral hernia is generally accepted today. The hernia enters a preformed pouch or diverticulum which has failed to become obliterated. In 200 consecutive post-mortem examinations reported by Murray, peritoneal femoral diverticula were found in fifty-two subjects; in fourteen of these, the sac was bilateral. Femoral diverticula were found more frequently

than inguinal diverticula; in the 200 subjects potential inguinal sacs were found in thirteen cases, and in four of these the sac was bilateral. Keith found femoral diverticula more frequently in adult subjects than Murray did.

Frequency of Femoral Hernia on the Right and Left Sides. Femoral hernia is usually single and appears most often on the right side. When it is bilateral, the hernia on the right side is usually the larger.

Age of Onset. Femoral hernia rarely occurs before the twentieth year. It is more frequent in women, becoming more common as age advances.

Anatomic Causes. The anatomic conditions that predispose to the development of femoral hernia are: a preformed sac; prominence of the peritoneal depression due to lack of fat; absence of the lymph gland of Rosenmüller; and a large saphenous opening, or a large compressible femoral vein. Joessel believed an open femoral ring an important factor.

Other predisposing causes are: pregnancy, parturition, emaciation, increased intra-abdominal tension due to obesity or tumors, and relaxation of the femoral ring in the aged.

Femoral Hernia Developing after the Operation for Inguinal Hernia. Femoral hernia occasionally appears after an operation for inguinal hernia. It is probable that the increased intra-abdominal pressure following the operation forces the omentum or intestine into a preformed femoral sac. Femoral hernia, like any other variety of hernia, may develop after any abdominal operation. When femoral hernia follows an operation for inguinal hernia, it is probably due to the pulling upward of Poupart's ligament by the deep sutures of the inguinal hernia operation which increases the size of the femoral opening. In operating for inguinal hernia it is sometimes advisable to stitch the conjoined tendon to Cooper's ligament when Poupart's ligament is lax or deficient, to prevent the secondary development of femoral hernia.

CONCLUSIONS

The present opinion is that except for the rare cases of traumatic hernia, all indirect inguinal hernias are congenital in origin regardless of the age of the subject when the hernia is first discovered. The presence of empty congenital sacs in adults is more common than is generally supposed. A majority of men undoubtedly carry these potential hernial sacs throughout life without evidence of hernia. Microscopic examination of the sac fails to show any difference between old and so-called new sacs.

Practically all physicians agree that indirect inguinal hernias are largely the result of a congenital or inborn condition or defect, which is gradually increasing, and that at most the injury complained of brought on the hernia a little sooner than it otherwise would have occurred. There usually is a weakened spot in the abdomen, and internal pressure has a tendency to cause a portion of the bowel to come through. Anything that causes internal pressure is likely to cause a hernia, sooner or later, in a person having a tendency to hernia. A strain such as a heavy lift or pull, or coughing or straining of any type may cause such internal pressure and bring about a hernia.

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Surgeons who treat fractures should familiarize themselves with the development of the lower humeral epiphyses and their appearance in roentgenograms. It is suggested that hospitals keep on file for quick reference *x*-rays of normal elbows at stated ages.

INJECTION VERSUS SURGERY IN THE CURE OF HERNIA

A CRITICAL SUMMARY

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IT was with some reluctance that I accepted the request to discuss the above subject because it has been my feeling, very definitely, that the injection method was not competitive to surgery as suggested in the title but should be considered as an alternative or supplemental method. However, when there is a certain element of choice between any two methods a comparative analysis seems indicated. This analysis will deal only with inguinal hernias since that is where the chief interest lies. The technique, with which the literature abounds, will not be discussed except in so far as it has a bearing on any particular phase of the subject considered.

OBJECTIVES SOUGHT FOR IN THE CURE OF HERNIA

The two objectives in curing a hernia are the eradication of the sac and strengthening of the wall (including, virtually, an obliteration of the inguinal canal).

Surgery. The high ligation and complete excision of the sac are the first requisites. Indeed, in young, muscular individuals with a small narrow-necked sac this procedure alone may be sufficient to obtain a cure in indirect hernia although, of course, the complete operation should be performed. The next procedure, that of strengthening the wall, becomes proportionately increased in importance depending on the weakness of the wall in any given instance. In the cure of direct hernias it becomes the most important feature of the operation, the treatment of the sac here varying between excision, puckering, plication, and simple reduction. The anatomic variations found are legion and the operative procedure should be varied to meet the individual case. The operator has no reason to restrict himself to the use of one or two stereotyped procedures. Flexibility in the operative attack is important to secure the best results in surgery. Results would be definitely improved if surgeons were more ready to modify the operation in accordance with the conditions found.

Surgery has the distinct advantage of laying the field bare so that the pathologic anatomy is exposed. The internal ring is constricted, the patency of the inguinal canal obliterated, the wall reinforced, and the course of the cord changed at will. After building a firm buttress to replace a weak wall the surgeon experiences a degree of immediate satisfaction that the user of the injection method does not feel. Hence, without considering the ultimate results, the surgical method offers the advantage of directly and immediately obtaining the objectives sought for in the cure of hernia.

Injection Method. Since the ideal extirpation of the sac cannot be accomplished, this method endeavors to obtain as a substitute: (1) an agglutination of the wall of the sac by adhesive inflammation and subsequent fibrosis; or (2) a fibrous constriction of the tissues around the neck of the sac so that abdominal contents cannot enter. Just how often either one of these developments occurs is speculative due to the insufficient number of patients who have been operated on following the injection treatment. It has been shown, however, that the sac usually becomes thickened and adherent to surrounding structures. Obliterated sacs have also been found, caused either by the chance injection of some of the fluid in the sac or, as is usual, by the effect of the contiguous chemical irritant causing a reaction in the serous surfaces and subsequent agglutination. Instances have also been found, however, where irreducible hernias have been

caused by injections, due to the formation of intrasacular adhesions firmly adherent to the hernial contents. Such an occurrence, of course, immediately precludes the cure of the hernia by the injection method. It is usually due to inefficient truss management. In obese individuals this complication is prone to occur and constitutes a definite drawback to injecting such individuals.

The ultimate fate of the sac in injected patients is not yet known. Some of them will doubtless reopen either partially or completely. At best, the fate of the sac in the injection method offers a rather poor comparison with the direct and complete excision by surgery.

The next principle, that of reinforcing the wall, is attempted by producing an aseptic inflammation with subsequent formation of fibrous tissue throughout the inguinal canal, constricting both the internal and external rings and, especially in direct hernias, building up tissue in Hesselbach's triangle. Another aim is to obliterate the inguinal canal by causing an agglutination of all the structures bounding it.

In a patient correctly managed the results obtained within eight weeks seem phenomonal to every beginner. The shrinkage in size of the external ring, the firmness of the tissues on palpation, and the absolute lack of an impulse on examination invariably instill great enthusiasm and optimism. But a patient who is given a series of injections deemed sufficient for that particular type of hernia, if no further injections are given and if the truss is discarded in six months, will usually show within the next year definite evidence of weakening of the wall, a moderate impulse, or a definite clinical recurrence. Partial absorption and stretching of the fibrous tissue is the cause of this and for this reason all experienced investigators agree that the patients must be overtreated initially; they must be reëxamined at intervals of one to two months when further reinforcing injections are given; and the truss must be maintained for a longer interval than was first thought necessary. In reality this prolongs the treatment for a period of perhaps eighteen months and may prolong it considerably beyond this. It may mean the retention of the truss for an indefinite period if the individual is active or engaged in labor. This is the view held by the more conservative proponents of the injection method particularly in direct hernias where there is a natural tendency for the tissue in Hesselbach's triangle to thin out progressively with the passing of time. This fact, superimposed upon the inevitable partial absorption of the newly formed fibrosis produced by injections, tends to cause a recurrence of bulging unless those two factors are counterbalanced by using additional injections and prolonging the use of the truss.

In the small indirect hernias in young, muscular individuals where a fortunate agglutination of the sac is obtained together with a constriction at the internal ring and a matting together of the tissues bounding the inguinal canal, a permanent cure is much more likely to be obtained.

As noted above, the injection method is, in certain respects, a blind procedure because the exact nature of the pathologic anatomy cannot be ascertained. Preoperative diagnosis is far from accurate and it is impossible in many instances to differentiate between a direct and indirect hernia. Many attempts at injection are doomed to failure by virtue of structural defects not apparent on external examination. Partially to obviate this, the injection method must handle all doubtful cases as though both a direct and indirect hernia coëxisted. Fortunately the milder solutions can be used in larger quantities and the placing of the needle need not be so exact. The fluid infiltrates the canal for a considerable distance. After the injection of the internal ring, a sufficient number of injections is given just beneath the external oblique aponeurosis along the entire course of the canal and over Hesselbach's triangle. This causes fewer complications and much less pain than if the solution is injected

more deeply at the very exact points above and below the cord as advocated by some (though perhaps seldom accomplished).

MORTALITY

Surgery. There is considerable statistical variation depending upon the age of the patient, type of operation, and other factors. Burdick, Gillespie, and Higginbotham reported 1,485 operations on 1,092 patients with a mortality of 2 per cent; Grace and Johnson reported 1,032 operations on patients over 50 with a mortality of 0.29 per cent; O'Shea reported 710 operations with a mortality of 0.7 per cent; and Watson collected a series of 8,000 operations with a mortality of 0.38 per cent. In general, the reported mortality may be said to average a little less than 1 per cent. This, of course, does not include the unreported mortality of less experienced surgeons in perhaps more poorly equipped hospitals where the mortality is undoubtedly much higher.

Injection Method. A few deaths have been reported, but in practically every instance they were caused by gross incompetence either in selection of cases or in the use of the older caustic solutions in doses far in excess of the amount that should be used. It is hardly any more fair to ascribe such instances to the injection method per se than it would be to penalize surgery because some tyro wantonly severed a contained loop of bowel in a hernial sac through gross inexperience or carelessness. With the use of the modern solutions and accepted technique the mortality of the injection method is practically nil.

Hence, although the mortality by operation is very low, it is definite, and the injection method offers a decided comparative advantage. It is impossible to evaluate how many additional recurrences by the injection method would be offset by a single operative death.

COMPLICATIONS

Surgery. Gibson and Felter reported 1,878 operations on 1,618 patients with about 388 complications (84 per cent of which were attributable to surgical trauma), such as hydrocele, 74; infected wounds, 70; hematomas, 59; pneumonia, 34; bronchitis, 32; coughs, 30; varicocele, 23; infectious diseases, 21; orchitis and epididymitis, 21; infarcts, 8; skin dermatitis, 6; tuberculosis, 5; influenza, 1; embolism, 1; empyema, 1; laryngitis, 1; spermatocele, 1. O'Shea reports 3.82 per cent deep wound infections in 1,016 operations for non-strangulated hernias. Fallis reports 1,600 hernia operations with complications developing in 116 patients. Certain complications which may be incidental to any surgical procedure, such as shock, tetanus, erysipelas, femoral thrombosis, etc., need no special consideration, but their menace is ever present.

Injection Method. Among the complications reported are pain, swelling of the cord, puncture of the peritoneum, hydrocele of the cord, drug sensitiveness, intravenous injection, hematoma, syncope, nerve injury, sterility (?), impotence (?), atrophy of a testicle (?), edema of the groin and scrotum, and certain complications incidental to truss wearing.

A few serious complications have been reported, such as cellulitis, sepsis, abscess formation, peritonitis, local gangrene, perirectal abscess, thrombosis of the deep epigastric artery, puncture of the iliac and femoral vessels, and fecal fistula. McKinney reported two cases of strangulated hernia occurring in 700 patients treated by injection.

Rice reports 1,020 patients, a total number including all patients treated irrespective of the final result or whether they continued or discontinued treatment, with 147 complications: induration of the spermatic cord, 72; skin erosion, 17; hydrocele, 17; general reaction, 16; coryza (due to tannic acid), 5; edema of the leg, 7; chemical peritonitis, 6; epididymitis, 1; dermatitis, 1; hematoma, 2; abscess, 2; gangrene of the bowel, 1.

There is very little evidence indicating the possibility of impotence and sterility as

sequelae of the injection method; indeed, experimental investigations by Rae indicate that these need not be feared and that there is very little likelihood of obliteration of the vas. The same applies to atrophy of the testicle. It should be borne in mind that from 8 to 10 per cent of scrotal hernias have atrophied testicles on the affected side.

With the use of the milder solutions, avoiding too deep an injection, immediate cessation of the injection if the patient complains of severe pain, with suction on the syringe before injecting to be sure that a vessel is not punctured and immediate withdrawal if blood shows in the syringe, and with careful examination at each injection to be sure that the hernia is completely reduced (particularly in a fleshy individual), complications can be kept down to a minimum and serious complications should be practically eliminated.

In my own experience, there has been no instance of a serious complication. Severe pain with mild shock occurred a few times, subsiding in a few hours without after effects. Blood appeared in the syringe on a few occasions but by promptly withdrawing the needle and applying pressure we made sure that no harm ensued. Subcutaneous ecchymosis has appeared twice. Indurated, tender, and swollen cords were seen occasionally, invariably subsiding spontaneously. Tender nodules beneath the external ring may appear if the fluid seeps through the ring. Annoying, persistent dermatitis on a few occasions has made truss wearing difficult. No instance of infection occurred. Indeed, the singular absence of complications has been noteworthy.

From the standpoint of complications, therefore, the injection method, by virtue of the rarity of serious disabling complications, offers a distinct advantage.

APPLICABILITY TO THE PROFESSION

The value of a method to the profession at large is a matter of considerable importance. The proportion of registered physicians who are qualified adequately to perform an operation for the radical cure of a hernia is relatively small. It requires a great deal more technical skill and training to qualify one to practice the operative method than it does to acquire the technique of the injection method. Nevertheless, the injection method has definite dangers if used indiscriminately, requiring training and experience, a requisite knowledge of anatomy, aseptic technique, and studious care. If these requisites are fulfilled the method should be in the reach of a much larger number of physicians than is the operative method.

APPLICABILITY TO THE PATIENT

It goes without saying that certain conditions such as strangulated, incarcerated, and irreducible hernias, and hernias complicated by undescended testes are contraindications to the injection method. Also, any hernia which cannot be kept completely and painlessly reduced by a truss is not applicable to treatment by injection (although some of these may be applicable for palliative injections if surgery is contraindicated or not used for some other reason). In addition, the injection method is contraindicated, as far as any hope for material success is concerned, in instances of ascites, large abdominal tumors, carcinoma, hemophilia, sliding hernia, true traumatic hernia (very rare), hernias in obese individuals, as well as those hernias in which the external ring is found to exceed 3 cm. in width. The duration and the size of a hernia are less important than the degree of defect in the wall.

The injection method may be considered as having favorable implications in the aged and infirm who are poor operative risks; for patients who have persistent fear and opposition to operation; for patients who, after being operated on, have a tendency to recurrence; in small postoperative recurrences where reduction can be effected; for small direct hernias on account of the relatively high operative recurrence rate; for moderate sized hernias where the external rings are not over 3 cm. in width; in instances of doubtful liability

in industry where the employer or insurance carrier may offer the method as a compromise because of the lessened cost involved; and for individuals with beginning weakening of the wall and enlarged rings in order to lessen the likelihood of a hernia developing, particularly to enable prospective employees in industry with such conditions to pass a pre-employment examination.

There is also a group of patients in which neither the surgical nor injection method can offer good prospects of cure, but in which, because of the lessened danger of the injection method, this procedure may be preferable. This also includes patients afflicted with prostatic hypertrophy, stricture, chronic bronchitis, obstinate constipation, tuberculosis, and certain other systemic afflictions. It is assumed, of course, that local pathology should be corrected if possible, but where this cannot be accomplished and where recurrence by any method is prone to occur the patient undergoes much less hardship by a partial post-injection recurrence than with a postoperative recurrence, and in addition, subjects himself to much less danger. Such post-injection recurrences, if they should develop, can often be kept under control by further injections.

From the foregoing it appears that except for certain definite and fixed contraindications, because of its inherent freedom from mortality and serious complications, the injection method has a rather wide range of applicability. However, it is again impossible to evaluate how much this increased range of applicability will compensate for the relatively higher recurrence rate in the injection method.

The treatment of hernias in the aged and infirm is a problem in itself. Many of these people are resigned to pass the rest of their days wearing a truss, but the truss becomes increasingly more unsatisfactory. Even though many such patients do not fulfil the important condition of a complete, painless reduction of the hernia by truss, they constitute a partial exception to this rule. If they are kept in bed for a week or ten days with a truss in position, a few injections being given during this period, and a series of subsequent injections given, the additional support furnished by the fibrous tissue reaction very often transforms an uncomfortable existence to a comfortable one and the hernia is kept under fairly good control even though not cured.

RECURRENCE

Naturally, in the comparison of results one of the most important criteria concerns the recurrence rate, and here we have to rely on statistics which, as the old saying goes, can be made to prove anything, even the truth.

Surgery. The surgical results vary, of course, depending upon whether the hernia is direct or indirect, the age and general condition of the patient, the size of the hernia, and the condition of the abdominal wall.

Indirect hernias show the most favorable results, the recurrence rate averaging from about 3 to 10 per cent. Erdman reports 665 operations at the New York Hospital with a recurrence of 3.10 per cent. Schwartz reported 207 examined from one to eleven years following operation by the Bassini method with a recurrence rate of 5.3 per cent. Ricketts collected a series of 6,027 operations performed by thirty-four surgeons with an average recurrence rate of 5.58 per cent. Fallis reports a recurrence rate of 7.4 per cent. Cowell reports a recurrence rate of from 3 to 8 per cent for direct hernias. Davis at the Massachusetts General Hospital traced 754 patients showing a recurrence rate of 8 per cent. Taylor analyzed a large series from the Johns Hopkins Hospital with a recurrence rate of 5.6 per cent.

Direct hernias show a much higher recurrence rate. Thus, in Erdman's series there was a recurrence rate of 16 per cent in 313 indirect hernias; Lameris reported 28.4 per cent in 102 patients; Fallis reported a 11.6 per cent recurrence; Taylor reported 18.08

per cent; Davis reported 15 per cent; and Andrews and Bissel, collecting 1,570 operations by eleven surgeons, showed an average of about 15 per cent recurring (the individual rate varying from 4 to 32 per cent).

Illustrating the poorer prognosis in elderly individuals, the series compiled by Grace and Johnson in patients over 50 years of age is significant in that the recurrence rate for indirect, single hernias was 18 per cent with a 25 per cent rate for double hernias; in those of the same age group operated on for recurrent indirect hernias, a recurrence formed again in 30 per cent, while in direct recurrent hernias a second recurrence occurred in 38 per cent.

In 1937 Burdick, Gillespie, and Higginbotham reported a series of 1,485 fascial suture operations in 1,092 patients at the Hospital for the Ruptured and Crippled. The mortality was 2 per cent. With the use of autogenous fascia there was a recurrence rate of 27.7 per cent; with homologous fascia the rate was 28.3 per cent; and with ox fascia the rate was 35.3 per cent. This series, of course, contained many unfavorable types, including direct hernias, combined direct and indirect, large scrotal hernias, recurrent hernias, and ventral hernias.

Wilmoth investigated 2,856 patients operated on and found that 6.3 per cent had been operated on before. Coley reviewed a series of 1,200 fascia lata operations and makes the statement that he prefers the use of silk. Druner reports a 21.4 per cent recurrence following the use of the Bassini method in a series of difficult operations. Nearly 10 per cent of Watson's private patients had had one or more previous operations.

Stanton estimates that in the hands of experienced surgeons in indirect hernias there will be a 5 per cent recurrence rate the first year with an additional 1 per cent recurrence each year thereafter. Direct hernias traced over five years showed a recurrence rate of 25 per cent.

There have been about 200 operative procedures advanced for the cure of hernia —which is somewhat indicative of the fact that the ultimate goal has not yet been attained. As the published recurrence rates are usually from qualified surgeons in well organized hospitals it is probable that the recurrence rate in the country at large is considerably higher. Relatively few surgeons have an accurate knowledge of their recurrences either because of failure to follow up their patients or because many patients with a recurrence fail to report back to the original operator.

Injection Method. There are several difficulties in accurately comparing statistical results between the surgical and injection methods. First, the operative method has been in use for so long that the results can be evaluated with a fairly high degree of accuracy. The modern type of injection therapy, however, has been in use by experienced operators for only about seven years, a period too short for any conclusion to be crystallized. A second difficulty is caused by a fact that a surgical procedure is completed at the time of the operation and any subsequent bulging is immediately classed as a recurrence; whereas in the injection method, since there is often no set time when the treatment can be said to be completed, and reinforcing injections are usually necessary after an elapsed interval, there is a tendency to interpret a subsequent bulging as a sign of unfinished treatment. When this bulging is corrected by the new series of injections, the interval following the last injections becomes shortened and the date of subsequent recurrence postponed. This is the only explanation I can give for the remarkably high percentage of cures reported by some.

Wollerman reports 94 per cent cured, Larson reports 93.5 per cent, and Harris and White report 95.9 per cent cured in what they interpreted the "good risk" group and 81.5 per cent in the "poor risk" group. Watson reported 94 per cent cured in small indirect hernias and 85 per cent in the large indirect type, with 80 per cent for

the large direct hernias. Rice, by means of a questionnaire sent to twenty-three physicians, collected 2,216 cases 85 per cent of which were cured, the average number of injections being eleven. There was no fatality in this group. Rice reported a cure of 97.6 per cent of the "controllable cases"; however, he states that ninety-seven patients either did not receive sufficient injections to cure the hernia or did not return for the final check-up. As any patient with a recurrence might be listed as not having had sufficient injection the final recurrence rate would doubtless be higher. Bratrud, who originally reported 96 per cent cured, more recently has reported 83 per cent cured. McMillan's first published statistics reported 92 per cent cured, but his subsequent reports have similarly shown a gradual increase in his recurrence rate. Quillin originally reported a recurrence rate of about 4 per cent, but has since made the statement that a recurrence will take place in a high percentage unless repeated series of injections are given and the truss worn for a long time. In marked contrast to other statistics is the report of Burdick and Coley with a recurrence rate of 83 per cent in fifty-six patients.

To aid in determining the outcome I studied one group of twenty-five patients given only one series of injections (the number of injections deemed sufficient for each individual case), in which the truss was worn for six months and then removed, no further injections being given. Within eighteen months a recurrence developed in almost half of the patients. Obviously, these patients were incompletely treated but, even so, the results of the study are significant. Various other physicians have reported similar experiences by personal communication.

Most physicians who have used the injection method for a number of years usually experience a definite cycle of changing impressions. The first feeling is one of great enthusiasm when, after about six weeks one feels the marked constriction of the external ring, the firmness of the tissues around the inguinal canal together with the patient's report that he no longer feels the hernia impinging against the truss when he coughs or strains; this enthusiasm is at its height after the series of injections has been completed and sufficient time has elapsed so that the truss can be removed temporarily and the patient is tested when he coughs and no impulse is felt. If, because of the combined optimism of the physician and the patient the truss is removed a few months later and left off, subsequent examination in the course of the next six to twelve months in most cases will show either a weakening of the wall or an impulse on coughing or both. When this occurs in a sufficient number of cases the physician is definitely disappointed if not discouraged.

To combat this it has become the rule to insist on a series of reinforcing injections every few months with a much longer period of truss wearing. Because of the tendency for some of this newly formed tissue to absorb and gradually to stretch and give way it is very difficult to estimate the result in patients five years after the last injections, assuming that no more injections are given during the interval. If it is the tendency for recurrence to follow one series of injections it is logical to assume that there will be a similar, though perhaps lessened tendency, for a recurrence to show up in subsequent series. Indeed, one is tempted to use the query "When is a recurrence not a recurrence?" because of the indefinite period of time consumed in treating a given case and the human tendency to reinject and to call a case one of unfinished treatment rather than a recurrence. For, strictly speaking, any subsequent bulging even though symptomless should be classed as a recurrence.

It is true, however, that recurrences following injections are usually much smaller than the original hernia and are frequently without symptoms. It is to be expected that such recurrences, if left untreated, will gradually grow in size. To avoid a high recurrence rate it is my belief that, except

in the most favorable cases, repeated series of injections will have to be given over a period of years with the truss worn almost indefinitely. Even if this should prove to be ultra-conservative it is far better for proponents of the injection method to emphasize some of the drawbacks inherent in the method than to report highly enthusiastic statistics which will not stand the test of time.

Should it develop in the future that the truss will have to be worn for a very protracted period in a high percentage of cases many will question any real need for the injections. The answer is that most patients experience a much greater degree of well-being and comfort from a combination of truss-wearing and injections than from the truss alone.

No one can deny that instances of brilliant cure by the injection method occur. When, after four years without truss wearing, no sign of hernia can be found the result obtained without operation must be classed as epoch-making. However, for every example of this kind, there are perhaps five who show some evidence of what should be classed as partial recurrence.

DISADVANTAGES OF THE INJECTION METHOD

In addition to the uncertainty as to a higher recurrence rate in the future there are other factors which will appear objectionable to certain patients. Although a well-fitting truss should not materially inconvenience a person, there are those who, although positively abhorring a truss, might be willing to wear it for a few months but if confronted with the probability of having to wear it indefinitely they very decidedly would prefer an operation. There are certain ones also who react similarly to undergoing a series of injections extending over a period of a year or more. A few individuals react unfavorably to repeated experience with the needle and to the varying degrees of discomfort experienced. Another difficulty lies in the fact that many patients will not follow the meticulous care in keeping the truss accurately applied—an absolute essential to success.

ADVANTAGES OF THE INJECTION METHOD

The relative safety and simplicity of the method have already been stressed and, of course, the ambulatory nature of the procedure is one of its greatest assets. The freedom from hospitalization and loss of time from work with the result of saving time and expense is a factor of greatest economic significance. This is of importance in industry today because of the increasing tendency toward pre-employment examinations and examinations during employment. Many corporations and insurance companies are averse to employing individuals with hernias. The use of the injection method and the truss offers the employee something which was not previously available. (The state laws vary in this respect; at the present time about twenty-two states give the patient a choice between the injection and operative methods, while twelve states insist upon surgery; other states advocate a cash settlement instead. The Compensation Department of the Works Progress Administration, however, does not recognize the injection treatment as a cure.)

The prophylactic value of the injection method should not be overlooked in those individuals with enlarged and lax rings and with perhaps a suspicion of an impulse on coughing. A series of injections may cause a sufficient alteration in both rings and canal to prevent the development of an indirect hernia. It is too early to be certain of this but the so-called potential hernia should offer a fertile field for future investigation by this method. A few injections a year serving to keep such cases under control would be an economic boon to a great mass of individuals.

SUMMARY AND CONCLUSIONS

After injecting and operating upon approximately equal numbers of patients

in the past four years, Earl states that he is operating on more patients and injecting less frequently as time goes on. Bancroft narrows the indication for the injection method down to those patients who refuse operation, those in whom the risk of operation is too great, and those with large hernias not held by a truss who either refuse operation or for whom an operation is contraindicated. In the latter group the injections reduce the discomfort by reducing the size of the hernia. He feels that the recurrence rate in injected hernias will be shown to be too great in the course of time. On the other hand, Bratrud, with a vast experience, remains quite highly enthusiastic and many others feel similarly as demonstrated by the statistical summary presented above.

No absolute and direct comparison between the injection and operative methods can be made because of the inherent difference in various factors which cannot be directly compared. Certain instances of contraindications to the one method can be used in the other and vice versa. The opinion is held that the injection method should be considered as an alternative to replace surgery in selected instances. From a cold analysis surgery is preferable because it is exact and definite and has a much lower recurrence rate. When one considers mortality, morbidity, expense, loss of time, and certain economic considerations, however, in all of which the advantages of the injection method are obvious, the latter method holds definite appeal.

The opinion is offered that the injection method holds its chief appeal in the following groups: first, in patients over 55 because of the higher mortality and morbidity by surgery; second, that group which includes both patients in whom operation is contraindicated and those belonging to the "poor risk" class; third, that group which for economic considerations finds the injection method preferable because of its ambulatory nature and consequent reduced cost.

It behooves the physician to be familiar with all aspects of the hernia problem so that he may give the patient the benefit of the latest information on this subject as regards both surgery and the injection method. The family of a patient who dies of some complication following a hernia operation might feel harshly toward a physician who did not inform them concerning the possibilities of the injection method. Likewise, a patient after receiving several series of injections only to have recurrence take place each time may not react favorably if the physician has not frankly discussed the possibilities with him originally.

Many surgeons condemn the injection method on theoretical grounds without having had any personal experience with it. On the other hand, many proponents of the injection treatment are much more optimistic than the results warrant. It remains to be seen whether the rising recurrence rate will be compensated by the advances in technique and the enhanced efficacy of the newer, improved (especially the tannin-containing) solutions.

The opinion is offered that surgery, in general, remains the method of choice. The injection treatment, however, offers a valuable addition to our armamentarium, to replace surgery in certain instances and to supplement it in others.

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REPAIR OF SEVERED TENDONS

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THE tendons most frequently injured are those of the hand, and therefore whenever the hand has been injured exact anatomic tests should be made for the function of each individual tendon. Inability to flex the terminal phalanx of the finger indicates division of the flexor profundus tendon; inability to flex the proximal interphalangeal joint indicates that the sublimis also has been divided; inability to flex the tip of the thumb shows that the flexor longus pollicis has been injured.

In addition to tests for the function of the tendons, the surgeon should also carefully test for division of the nerves which lie in close proximity to the tendons. This is of extreme importance since tendon repair does not proceed satisfactorily in the presence of a damaged nerve. Remember that each finger has four digital nerves whose distribution can be charted with a fair degree of accuracy. Injuries to the median and ulnar can be easily diagnosed because of their exact distribution. In several hundreds of cases of ulnar nerve injury, variation from the normal cutaneous distribution has been found in less than 1 per cent. It almost invariably supplies sensation to the little finger and to the ulnar half of the ring finger. If injury has occurred below the point of origin of the dorsal branch of the ulnar nerve, the palmar half of the fingers is involved; if above the dorsal branch, both the dorsal and the palmar surfaces are involved.

Having diagnosed the division of a tendon, the surgeon's next problem is to decide whether to suture the severed tendon at once or to postpone operation. This decision requires experience and nice judgment. Wounds caused by clean glass or by sharp instruments such as razors or knives should usually be sutured at once. Milk bottle injuries are almost always infected and primary suture is contraindicated. Soiled wounds containing dirt or other foreign bodies may sometimes, after thorough cleansing, be successfully sutured. In general it is a wise policy to perform the primary suture even though there is a possibility of suppuration.

Irrespective of whether the suture be performed at once or delayed, there are certain principles of tendon surgery which must be strictly adhered to. These principles are based on the anatomy and physiology of tendons. Tendons are easily traumatized, even more easily than nerves are, and therefore a nontraumatic technique is indispensible. The surface of the tendon is coated with a layer of differentiated cells whose nuclei are round instead of elongated like the typical tendon cells. It is these cells which give the peculiar smoothness and glisten to the surface of the tendon. If the tendon is injured by being grasped with a mouse-tooth forceps, an irregularity results, with a tendency to adhesion at that point.

The tendon is coated with a layer of loose connective tissue called paratenon. This is rich in elastic fibers and is an essential element in the free gliding of the tendon. It is important to conserve this tissue wherever possible and not to strip it off the tendon. Within the tendon sheath the paratenon is not present except on that surface of the tendon which is not exposed to friction. The sheath is found wherever a tendon has to change its direction; its function is to diminish friction by acting as a fluid buffer. Its surface is lined with a layer of flat cells which provide its peculiar

smooth appearance. Like the surface of the tendon, these cells are easily injured and if traumatized, tend to adhere to the tendon.

that even experienced surgeons are guilty of mistaking one for the other. I am inserting a simple diagram of the arrangement of

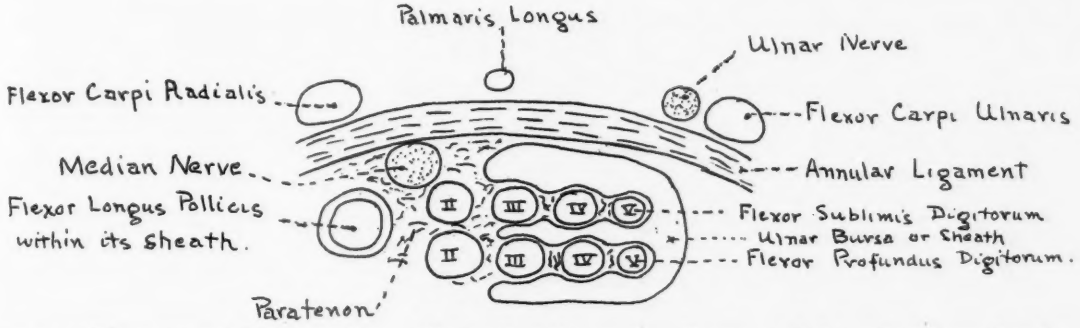

Fig. 1. Diagrammatic cross section at the level of the annular ligament of the wrist illustrating the relationship of the tendons to one another and to the nerves.

Because of these anatomic peculiarities, the severed tendon should be grasped only at its divided end and not more should be traumatized here than is going to be sacrificed at the time of the tendon suture, namely, about 1/16 to 1/8 inch. Traumatism to the tendon, the paratenon or to the sheath almost invariably spells disaster; success is difficult enough even with accurate atraumatic technique.

A second important fact to remember is the rule of tendon tension. If a tendon such as the flexor carpi radialis is divided, it will be found that under deep anesthesia the tendon ends will approximate accurately if the wrist is flexed to the limit. If, on the other hand, the wrist is extended, the ends will separate one or more inches. In suturing a tendon, therefore, an attempt must always be made to restore the normal degree of tension. This means that a maximum of 1/4 inch of tendon may be sacrificed. If more is removed there will be excessive tension and a contracture is liable to develop.

If the tendon division occurs at the wrist, accurate knowledge of the anatomic arrangement of the tendons and nerves is essential. In a surprising percentage of reoperations I have found the proximal end of a tendon sutured to the distal end of a nerve or the wrong tendons sutured to one another. It may seem to the uninitiated an easy matter to distinguish severed tendon from severed nerve, yet the fact remains

the tendons and nerves at the wrist. (Fig. 1.) This diagram should be memorized by every surgeon who intends doing tendon work on the hand. By strict adherence to the anatomic arrangement it is possible to solve the puzzle of nine divided finger tendons, three wrist tendons and two divided nerves. Without some such guide the surgeon is lost.

When both the flexor sublimis and profundus tendons have been divided within the digital sheath or close to it, I advise suturing only the profundus tendon. If the sublimis is sutured also, it is likely to become adherent to the profundus and thus rob the profundus of its action on the terminal phalanx.

PRIMARY TENDON SUTURE

Within a few minutes after the division of a tendon, the ends retract. The only exception to this rule is in those cases in which the division of the tendon is incomplete or if an emergency splint has been applied at once holding the joint in such a position as to give complete muscle relaxation. If the cut extends across the proximal phalanx of the fingers, the distal stump of the flexor profundus will be found to be only 3/4 inch long; the proximal stump will usually have retracted about 2 inches. Realizing these facts in advance, the surgeon plans his incision accordingly. It is a waste of time to "fish around" in the

wound itself for the divided stumps. This merely traumatizes and increases the risk of failure. Small incisions should be made at the points where the surgeon expects to find the stumps of the tendon. These incisions should be placed preferably in the normal creases of the skin; they should never cut the crease at a right angle. They must also be so placed as to avoid injury to the sensory nerves. It is my habit after dividing the skin to proceed with blunt dissection until the delicate digital nerves have been exposed; then the knife may be used again.

Fig. 2. Shows range of motion following secondary tendon suture for division of the flexor sublimis and profundus tendons of the index, middle and ring fingers. Secondary suture was performed three months after an attempted primary suture had failed.

After the stumps have been found the end of the tendon should be grasped delicately with a clamp. My own preference is for a small vulsellum forceps of my own design, but a mosquito hemostat will do if carefully used. Do not grasp the surface of the tendon with a mouse-tooth forceps or with any other toothed instrument. For suture material I advise braided Deknatel silk. Each piece should be tested in advance both by the suture nurse and by the surgeon to be sure that it is of sufficient strength to withstand a strong pull. The best suture in my experience is the one devised by Dr. Bunnell of San Francisco and myself. This is inserted by means of two fine straight round needles, preferably calyx-eyed. If no calyx-eyed needles are available one may use the ordinary cambric needle, which should always be threaded before boiling. A needle is attached to each end of a piece of silk 10 inches long; the size of the silk varies, depending upon the size of the tendon. If Deknatel silk is used, No. 2 is usually right for the adult flexor profundus tendon. The stitch is inserted as follows: The operator, grasping the tendon clamp, steadies the tip of the tendon against the index finger of the left hand, and with one needle transfixes the tendon horizontally ¼ inch from the severed end. The needle then traverses the tendon

obliquely at an angle of 45 degrees, emerging on one side, and is then reinserted exactly in an opposite direction to the other side of the tendon. Two oblique stitches are taken with the second needle corresponding to the two taken with the first. The clamp is then removed and the tendon steadied by holding on to the two silk threads. The traumatized tip of the tendon is removed with a very sharp knife or sharp scissors so that a clean cross-cut of tendon is visible. One additional stitch is then taken with each needle, bringing the silk stitches out as near the center of the tendon as is possible. Traction of 2 to 3 pounds is put on the silk so as to take in all the slack and bury the silk in the substance of the tendon. The same stitch is taken in both tendon ends.

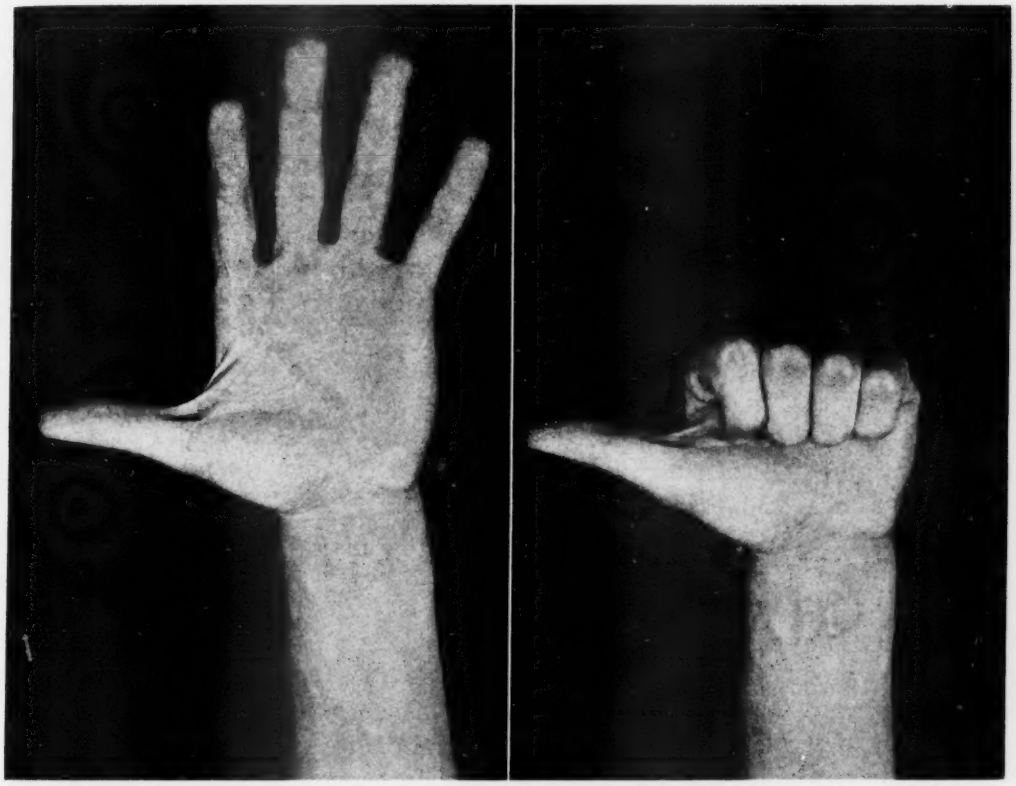

Fig. 3. Shows the range of motion of the index finger following complete division of the flexor sublimis and profundus at their entry into the theca. Secondary operation done nine years after the original injury. Tendon sheath reconstructed by the celloidin tube method. The proximal stump of the flexor profundus was lengthened by means of a free graft taken from the flexor sublimis of the same finger.

To approximate the tendons a probe is next passed along the course of the tendon. If this be in the finger the probe will pass through the tendon sheath, if in the palm care must be taken to see that the probe does not damage the nerves in its course. The ends of the silk are then threaded into the eye of the probe and the two tendon ends are drawn down into the original wound. Each strand of silk is tied separately to the corresponding strand of the opposite tendon end. In tying these knots accuracy is essential. Sufficient tension must be applied to cause the tendon ends to buckle a trifle. The knots must be squared to avoid possible giving. Three turns should always be taken. When the suture has been completed the two tendon ends ought to lie in perfect apposition, the silk knots buried between the tendons so as to be invisible. No silk should show on the surface of the tendon at any point. There should be no raw surface of tendon exposed at the suture line. To perform this suture accurately requires much practice and

since few surgeons have occasion to perform a sufficient number of tendon sutures, this practice should be done on the cadaver. When working on the tendons of the cadaver it is a good plan to test the accuracy of the operation by removing the sutured tendons and applying traction to each end. A good suture line will withstand a force of 5 to 10 pounds without separation of the tendon ends.

The soft parts should be closed with accuracy, great care again being taken not to include the digital nerves in the suture. A superficial drain may be used but should be removed within forty-eight hours. The part should be immobilized either with plaster of Paris or a splint in such a position as to take the tension off the divided tendon. The after treatment will be considered later.

SECONDARY SUTURE OF DIVIDED TENDON

This procedure is applied to those cases in which the surgeon temporarily postpones operation and also the numerous cases in which, despite primary suture, separation of the tendon ends has occurred either because of incorrect technique or suppuration. The problems involved in secondary suture are far more complex than when the primary suture is possible, for here the surgeon is dealing not only with the division of the tendon but also with a shortening of the tendon due to the retraction of the ends. Since secondary suture usually has to be postponed many weeks after the primary injury, this retraction amounts in most instances to 2 or more inches. Such a gap between the tendon ends cannot be overcome by traction and therefore some type of tendon transplantation must be employed. Two methods are available, either the pedunculated transplant or the free transplant. The pedunculated transplant is applicable to those cases in which there is a nearby tendon which can be sacrificed to replace the divided tendon. For instance, in the palm an adjacent flexor sublimis tendon can be sacrificed to replace the divided flexor profundus tendon. On the dorsum of the hand the extensor indicis proprius may be utilized to replace the extensor of the thumb, the middle or even of the ring finger. If the division of the tendon has occurred within the theca and the finger, the pedunculated method is not applicable and a free graft has to be used. Frequently in the palm it is advisable to use a free graft rather than the pedunculated method. The technique of the operation varies considerably, depending upon the anatomic site of division of the tendon. I shall take up first the simpler type, namely, the tendon division in the palm where either the pedunculated graft or the free graft may be used.

THE PEDUNCULATED GRAFT

I will assume that the divided tendons are the flexor sublimis and profundus of the middle finger, and that division has occurred approximately in the middle of the palm. The first incision is made near the base of the middle finger following the oblique crease which usually passes between it and the index finger. With careful avoidance of the digital nerve, the tendon sheath is exposed and the tendons identified. Often the flexor profundus will be found fused to the flexor sublimis. If this has occurred the profundus should be carefully separated from the sublimis. In this situation the profundus lies deep to the sublimis tendons which have separated into their two halves. The tendon stumps are as a rule rounded over and smooth. The tip of the profundus tendon is grasped with a tendon forceps, the typical tendon stitch is inserted and the rounded end of the tendon cut off so as to expose a clean cross-cut. The first incision is temporarily closed so as to avoid drying out of the tendon. The second incision, about 1½ inches long, follows the course of the flexor tendons of the index finger. The tendons are exposed in the palm and the flexor sublimis followed down to its entry into the digital sheath. With a probe the length of tendon required to meet the distal stump of the flexor profundus is

measured off. The tendon is cut across at this point, the end grasped with the tendon clamp and the usual stitch inserted. An oblique tunnel is bored from the course of the index finger tendons at the proximal end of the incision over to the distal stump of the divided flexor profundus of the middle finger. The flexor sublimis tendon of the index finger is drawn along this course until it lies in apposition with the divided tendon of the middle finger. The knots are then tied exactly as in the primary suture.

It is a great aid in determining the accurate tension if the operation is done under a local anesthesia. At the conclusion of the operation the patient ought to be able to extend the finger completely without separation of the tendon ends and be able to flex it almost to the normal limit. The wounds are closed in the usual fashion. It is obvious that in this operation the sublimis tendon of the index finger is sacrificed. Although this loss does not seem to be noted by the patient, it is the disadvantage of the pedunculated method and I therefore usually favor the free tendon graft which does not necessitate the sacrifice.

THE FREE GRAFT METHOD

The first incision corresponds exactly with that for the pedunculated operation and the usual stitch is inserted into the distal stump of the flexor profundus. The second incision extends in a proximal direction along the course of the proximal stumps of the divided flexor tendons. As a rule the carpal sheath of the tendons has to be opened up so as to give adequate exposure of the proximal portion of the flexor sublimis and the flexor profundus. The two stumps have generally fused to one another and should be carefully separated exactly in the line of cleavage. The flexor profundus is then threaded with the Bunnell-Mayer suture and a corresponding suture is taken in the flexor sublimis stump. With a probe, the distance between the proximal and distal flexor profundus stumps is measured and this distance marked off on the flexor sublimis. The flexor sublimis is cut across at this level and the part thus removed is spliced into the gap between the two flexor profundus stumps. It is advisable to insert a portion of the tendon stitch before cutting away the flexor sublimis graft. It is scarcely necessary to add that if accuracy of suture is necessary for the primary tendon suture, even greater accuracy is required for the free tendon graft. Here the vitality of the tendon depends upon absolute accuracy of coaptation and even the slightest inaccuracy spells failure.

THE BUTTON-HOLE STITCH

On the dorsum of the hand the tendons are usually too flat to permit accurate end-to-end approximation. It is therefore wiser to use the button-hole overlapping method of suture rather than the end-to-end stitch. As an example, assume the division of the extensor tendon of the middle finger 1 inch proximal to the metacarpophalangeal joint. The operator has decided to utilize one of the two extensor tendons of the index finger as a substitute for the divided extensor of the middle finger. The first incision, slightly convex, exposes the distal stump of the divided extensor. The end is usually bound down by delicate adhesions. The tendon is freed for a distance of about ½ inch, its end is grasped with a tendon clamp and with a No. 11 Bard Parker blade a slit is made in the tendon ¼ inch from its divided end. The incision is temporarily closed. A second incision follows the course of the extensor indicis tendon and is about 1½ to 2 inches long. Either the extensor indicis or the branch of the extensor communis of the index finger may be used as the transplant. The tendons are carefully separated from one another and one of them is cut across just proximal to the metacarpophalangeal joint. It is drawn through a subcutaneous channel to the distal stump of the divided middle finger extensor. A mosquito clamp, run through the slit in the divided extensor

tendon, grasps the tip of the extensor indicis and pulls this through the slit. There should be an overlap of the two tendons of about ¼ inch on each side of the slit. The suture is inserted by using a finer Deknatel silk than in the end-to-end suture since the strain does not fall directly on the silk strand. The first stitch passes directly over the chiasm of the two tendons, it perforates the substance of the slit tendon then passes through the replacing tendon, then through the other half of the slit tendon. The same needle then reverses its direction coming out 1/16 inch from its point of entry. Only one needle is used in this stitch. The knot is tied, leaving a small protrusion of silk on the side of the tendon where adhesion is less likely to develop. Two additional similar stitches are taken distal to the chiasm and two proximal to it. The two end stitches should be taken in such a way as to bury the projecting tendon end as accurately as possible. Complete coaptation is impossible, but on the dorsum of the hand risk of adhesion is so much less than on the palmar surface that slight inaccuracy may be permitted without endangering the result.

RECONSTRUCTION OF TENDONS WHEN THE DIVISION HAS OCCURRED WITHIN THE DIGITAL THECA

When tendons have been divided within the sheath of the finger, their reconstruction is peculiarly difficult. This is due to the fact that the normal theca is a narrow passage which just admits the passage of the tendons. Second, because after a division of a tendon, even if no infection has occurred, the sheath degenerates so that its smooth wall disappears and the tendon stumps become bound to it by adhesions. If a mild grade of infection has occurred, as is frequent, these adhesions become extremely dense, completely destroying the gliding mechanism of both tendon and sheath. Even if a free graft is accurately inserted it will almost surely become adherent to its surroundings because of the presence of these adhesions. To prevent this some surgeons have had recourse to the construction of an artificial tendon sheath by means of the tunica vaginalis of the testis.

My own attempts to solve the problem have been described by Dr. Ransohoff and myself in previous articles. The method we have used is the celloidin tube implantation technique. This method has as its principle the peculiar fact demonstrated by Dr. Frederick Prime, Jr. of Columbia University, that when a tube of chemically pure celloidin is imbedded in the human body, instead of exciting the usual foreign body tissue reaction, it becomes coated with a layer of delicate flat connective tissue cells which microscopically as well as macroscopically closely resemble the cells lining a tendon sheath. The method was originally utilized in a research to prevent adhesions between the brain and the skull after extensive resection of the dura. Ransohoff suggested that a celloidin tube might be utilized to develop a smooth-lined sheath after the digital sheath had been destroyed by trauma or infection.

The operation must be done in two stages. At the *first stage* the celloidin tube is implanted, at the second stage four or five weeks later, it is removed and the substituting tendon is drawn downward through the smooth tunnel left by the withdrawal of the celloidin tube. The details are as follows: An incision is made along the lateral border of the finger, permitting the excision of the scarred tendons and sheath. In most cases the two tendons are completely fused and so firmly adherent to the walls of the sheath that all motion is impossible. The excision must extend in a proximal direction as far as the stumps of the tendons. In some cases it has been possible to avoid this extensive incision by tunneling into the finger through a small transverse incision at its base. This method is applicable when there has also been an extensive sloughing of the tendons so that an actual removal of scarred tendon tissue is not necessary. In either event, whether incision be used or the tunneling method, there must be a

sufficiently large opening in the finger to permit the insertion of a celloidin tube. The tubes are made in three sizes, 6, 8 and 10 mm. in diameter. A tube of appropriate size should be used. One extremity must extend to the distal insertion of the flexor profundus tendon, the other end to the proximal stump. The superficial tissues are closed carefully over the celloidin tube. It is essential that the tube should at no point be covered only by skin. There must always be a layer of subcutaneous tissue and preferably a deep layer also. The finger is splinted and the wound allowed to heal. Experiments have shown that after four weeks the tube is surrounded by a layer of well-formed flattened connective tissue cells. At the expiration of this period the patient is ready to enter the hospital for the second stage.

Second Stage. The first incision is made near the insertion of the flexor profundus tendon and the tip of the tube identified. A second incision is made in the palm near the proximal portion of the tube. When the tube has been exposed, a probe threaded with a guide suture of silk is passed through it from one end to the other and the tube is withdrawn leaving the guide suture running down through the artificially constructed sheath. It has been most gratifying to note that in all cases the lining membrane of the channel thus formed is smooth and corresponds accurately in appearance with the normal tendon sheath. The proximal stump of the profundus tendon is exposed, threaded with a tendon suture exactly as in doing a free graft implantation. A free graft of the sublimis tendon is next removed or, if the sublimis tendon is not available, some other suitable substituting tendon should be used. To remove this free graft an incision almost always has to be made above the wrist. This should never cut across the transverse crease of the wrist since if this is done keloid formation is likely to result. The graft threaded at both ends is drawn downward through the newly constructed sheath of the finger by means of the guide suture. Its proximal portion is sutured to the proximal stump of the divided flexor profundus, its distal portion is drawn through a small drill hole made through the base of the distal phalanx. To draw the stump through this channel a ¼ inch dorsal incision has to be made running between the base of the nail and the distal interphalangeal joint. Great care must be taken here not to damage the matrix of the nail.

AFTER TREATMENT

The same principles apply both to primary and secondary suture. Although it might seem that motion should be begun immediately after operation so as to avoid adhesions, my experience has been that this is a dangerous procedure. There is too much chance of the tendon ends separating or of a failure of union of the wound. I have found it wiser to keep the finger immobilized until the wound edges are firmly united. This takes as a rule seven to nine days. As soon as union has occurred it is safe to begin with gentle exercises. No force should be used to straighten the joints which have been immobilized in a position of some flexion. I have found it advisable to supplement active contraction of the muscle by electrical stimulation of the involved muscles. As the electrical current causes contraction of the muscle the patient is instructed to attempt to move the injured part. At first the result is almost always disappointing and the range of motion is pitifully slight. Great patience and persistence are required to restore motion to the injured tendons. The surgeon must attend to this portion of the work himself or, if he delegates it to a physiotherapeutic aide, she must be carefully instructed in the exact details of the treatment. Failure is certain to occur if this important part of the treatment is left to an inexperienced assistant. To perform a tendon suture and then allow the patient to return to some distant home is a serious mistake on the part of the surgeon. Patients must be instructed at the outset that a lengthy period of postoperative treatment

is necessary and that unless this can be carried out the operation is contraindicated.

CONCLUSIONS

Reconstruction of severed tendons constitutes one of the most delicate problems in surgery—a challenge to the ingenuity and dexterity of the operator.

The difficulty of the procedure is illustrated by a remark made to the author by Alexis Carrell; this master of surgical technique said that he considered the restoration of function to damaged finger tendons the most difficult of surgical problems.

In this article I have attempted to give only a few of the details of mending divided tendons. It is hoped that it will serve as an incentive to further study on the part of many other surgeons.

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A NEW CONCEPT OF TENOSYNOVITIS AND THE PATHOLOGY OF PHYSIOLOGIC EFFORT*

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IN traumatic surgery it has been customary to consider trauma as occurring suddenly and with violence. In a rough physical sense the momentum (mass times the velocity) of the injuring object or falling body expends its force suddenly against the inertia of the stationary human frame or the stable surface upon which that human body falls. When these forces exceed the limits of elasticity of the tissues of our anatomic structures, fracture of bone, rupture of tendon or muscle or other soft tissue takes place.

The expenditure of such energy upon a large surface, in addition to exceeding the elastic limits of body tissues, will cause contusion trauma up to the point of death of tissue (necrosis of skin, subcutaneous fat, or the organism as a whole). Lesser degrees of energy expended against a very narrow surface or point, produce the incised and puncture wounds commonly met in practice.

Since all such accidents and inflicted trauma take place almost instantaneously, we have always disregarded the time element. Although the time interval through which such forces acted might vary, the important consideration has been the mass and velocity reacting against the aforesaid inertia. Naturally, forces involved have been unphysiologic, and often beyond the energy expenditure in the same moment of time by the coördinated physiologic efforts of our bodies.

The normal forces expended in physiologic movement of muscles and tendons, however, can and do produce pathologic alterations of both the acute and chronic type. The essential external feature differentiating these lesions from the acute trauma of violence is the time factor over which the forces are exerted.

Crepitating peritendinitis is an example of an acute pathologic lesion resulting from the prolonged exertion of unaccustomed muscular effort.[1] This lesion is recognized by the appearance of pain and crepitation on motion of a particular part of an extremity. Swelling, local heat and redness may be present over the musculotendinous junction of the muscle group involved. The crepitation can be felt or heard on movement of the tender part. Active contraction of the involved muscles, as well as passive stretching produces acute pain.

Crepitating peritendinitis develops from certain general predisposing factors. In the normally sedentary adult, the prolonged and continued muscular effort of a certain task, sport or hobby, involving particular muscle groups, may be followed by the onset of mild or severe forms of crepitating peritendinitis in the muscle groups, subjected to that unaccustomed exertion.

Such examples are found in the professor of medicine, who weeds the garden, prunes the hedge, and spades the flower border on a bright spring morning. The onset of peritendinitis crepitans in the right forearm the following days confirms his distaste for bodily labor, and furnishes the alibi for his peaceful retirement to the fireside or study in spite of his connubial partner's future protests on the state of the shrubbery. An insurance executive went deer hunting. Ten days of scrambling hill climbing produced fatigue in the calf and thigh muscles. On the evening of his return home, his wife, having remained by the fireside,

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insisted on going dancing. This was the last straw. The following morning, he had crepitating peritendinitis of the tendo Achilles.

oped crepitating peritendinitis. (Dr. Martin Debenham, personal communication.)

Peritendinitis crepitans is frequently initiated by blunt gross trauma. In such

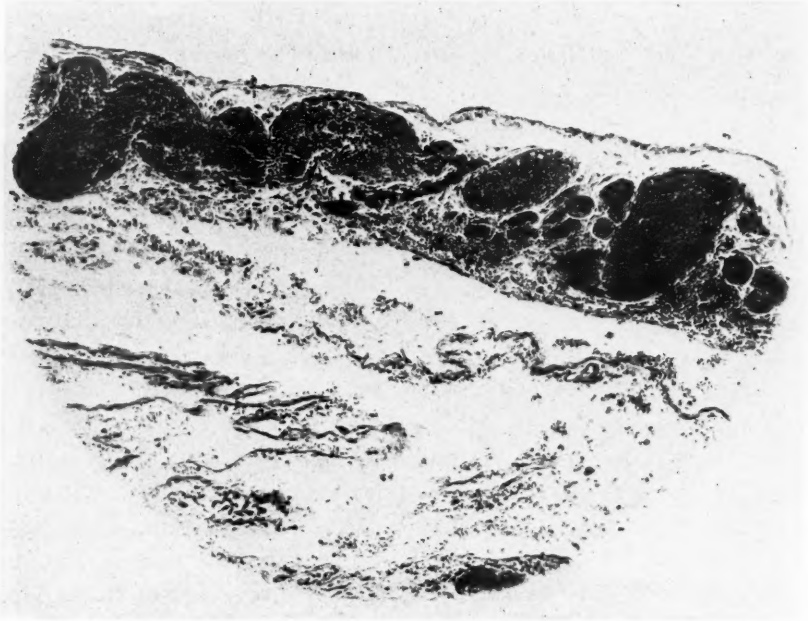

FIG. 1. Photomicrograph of crepitating peritendinitis under low power. Thrombosis of venules in the edematous peritendinous areolar tissue.

Inactivity—a long "lay off" from a particular job—can be followed in the same way by peritendinitis crepitans in a certain muscle group when the reëmployment involves use at high speed over a long period of time. This was especially made evident at the start of the N.R.A. reëmployment (not W.P.A.) by the increase in the number of such cases. Normal and accustomed labor, if prolonged, speeded up, or the muscle tendon excursion increased may result in the same disease process, even to a small "epidemic" occurrence. In a manufacturing plant making flashlight batteries, few employes developed crepitating peritendinitis over the course of several years. At the time of the 1931 Shanghai incident, flashlights in large quantities were ordered for the Chinese Nineteenth Route Army. The workers in the factory worked longer and faster, and, in wrapping labels on the batteries, they had to increase the excursion of the wrist movement, for the batteries ordered were not standard size. In one week, fifteen employees developed crepitating peritendinitis. (Dr. Martin Debenham, personal communication.)

Peritendinitis crepitans is frequently initiated by blunt gross trauma. In such instances, the workman continues the normal and accustomed task after the receipt of trauma, and hours or days afterward he may develop a typical crepitating lesion. A night watchman on his round, walked eighteen to twenty miles each night. One evening, he struck the right leg against a projecting pipe, fell and sustained a bruise and abrasion over the belly of the tibialis anticus muscle. He continued his rounds that night and the following night, before peritendinitis crepitans of the dorsi flexor group of muscles of the leg appeared.

About one-half the cases personally studied have been associated with trauma followed by usual exertion or toil. One must distinguish between trauma during employment followed by exertion while pursuing a hobby or sport, and the converse. A janitor, while helping move a piano, caught the right forearm between the piano and the door frame, sustaining a contusion and bruise. Five days later he reported to the industrial physician with crepitating peritendinitis of the forearm, radial extensor

muscle group. Further questioning brought out that on Saturday and Sunday (the days following the accident) he had for the first time made a model boat, gouging out the hull from a single block, with a small straight chisel. His painful symptoms appeared Sunday afternoon. Without the unusual effort of fine, rapid muscle movement, the condition would not have arisen.

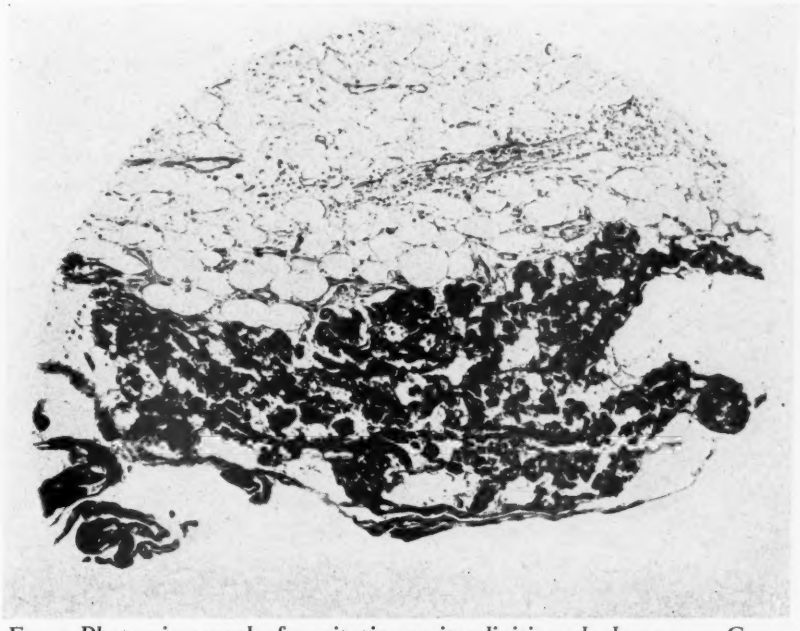

Fig. 2. Photomicrograph of crepitating peritendinitis under low power. Coarse masses of fibrin (stained black with Weigert's fibrin stain) in the peritendinous areolar tissue.

The pathologic changes accompaning crepitating peritendinitis have recently been described.[1] Briefly they consist of edema of the peritendinous areolar tissue and muscle, particularly the muscle tendon junction. Thrombosis of the venules occurs in these tissues. (Fig. 1.) Deposits of fibrin in the areolar tissue (Fig. 2) and between muscle fibers are found to be the cause of the audible and palpable crepitation. The muscle may undergo degenerative changes to the point of liquefactive necrosis within the sarcolemmal sheath. (Fig. 3.) The areolar tissue and muscle both are found to have a more acid reaction, when colorimetric hydrogen ion concentration studies are carried out. The individual muscle fibers lose their glycogen, and such tissue extracts are found positive for lactic acid.

Both grossly and microscopically, the tendons are found unchanged. No tendon sheaths exist at the site of pain and swelling, and they cannot be involved in pathologic changes described above. Synovial lined spaces, peritoneum, pleura, and joint react to irritation in an entirely different and typical way. The "crepitating tendo vaginitis" is a peritendinitis and myositis, the product of excessive fatigue of a definite muscle group, and not of rheumatoid, infectious or toxic origin.

In a study of seventy-two cases of crepitating peritendinitis, it was found that 91.5 per cent occurred in males and 8.5 in women (adding to thirty-two cases previously described[1]). Thirty-eight cases followed direct trauma and accustomed or usual work and activity. The interval between the direct trauma and the development of crepitating peritendinitis averaged three days, with the shortest period one day. Thirty-four patients gave no history and showed no evidence of direct trauma or sprain, and all had either returned to work or sport hobbies after long inactivity, or had unusual tasks or unaccustomed exertion assigned to them. The muscle

tendon groups involved are shown in Table 1.

Muscle fatigue is a well known physiologic phenomenon and was extensively investigated twenty or more years ago. The essential features were firmly established and the chemical nature of the fatigue reaction entered upon at a time when the chemical knowledge and technique would not permit further elaboration. There is definite need for a further study of the chemical and physiochemical reactions in muscular fatigue, especially since new factors have been discovered in the chemical reaction of normal muscular contraction.[2]

In physiologic experiments fatigue of a muscle group is shown by more or less complete loss of irritability and contractility brought on by functional activity.[3] When complete fatigue occurs, a very long interval is necessary for recovery. After complete fatigue has occurred, further efforts to contract the muscle greatly prolong the period of recovery.[4] In crepitating peritendinitis, the loss of irritability and contractility of muscle involved is well shown on operative exposure of the muscle.[1] Most patients complain of a feeling of "loss of power" entirely apart from the pain experienced.

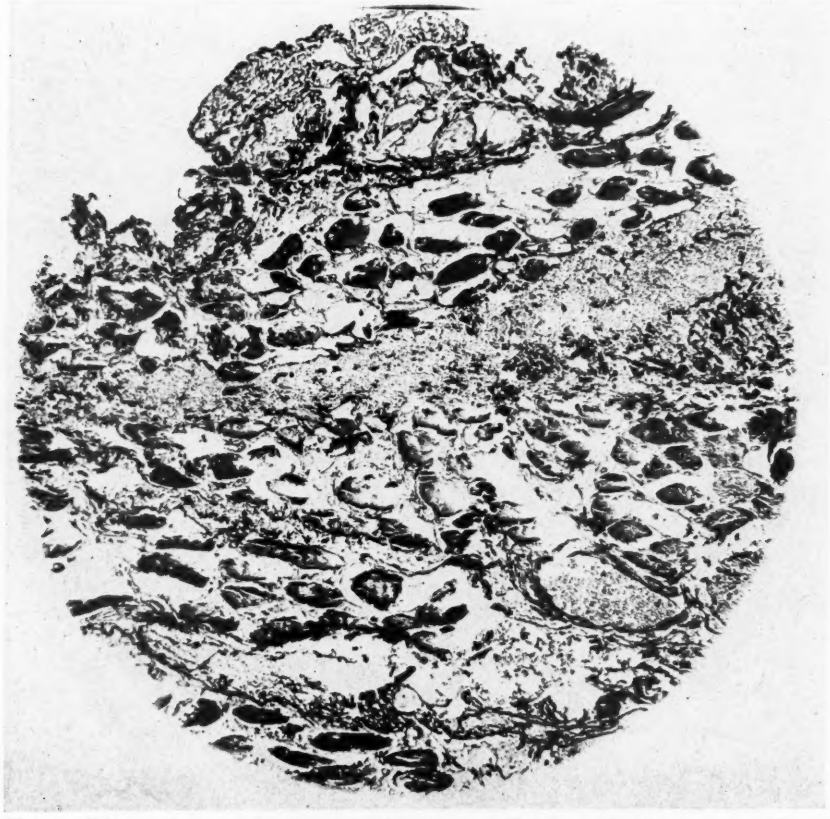

FIG. 3. Photomicrograph of crepitating peritendinitis under low power. Shows the liquefactive necrosis of muscle fibers, the interstitial edema and blood extravasation, and the coarse fibrin deposits in the upper and mid portion.

Treatment logically should be directed towards complete and absolute rest of the involved muscle group. At first glance, this seems to need no emphasis. A frequent mistake is made in immobilization of the wrist, or wrist and fingers, leaving the thumb free. If the thumb abductor longus and extensor pollicus brevis are involved, as they usually are, in connection with the two radial carpal extensors, such immobilization is inadequate. The thumb should be included in the plaster splint, held in a comfortable position of function. The examining physician may find on firmly

fixing the wrist and thumb with his hands, that no pain or crepitation occurs on movements of the fingers. The fingers then may be left free. However, when fixation of the wrist fails to prevent pain and crepitation on movement of the fingers, the digits must be immobilized in the plaster splint as well. Moulded plaster of Paris splints, lightly reinforced by circular plaster seem to be most effective and convenient in treatment of forearm and leg lesions.

TABLE I

MUSCLE TENDON GROUPS INVOLVED IN CREPITATING PERITENDINITIS*

Muscle	Cases
Extensor carpi radialis longus	37
Extensor carpi radialis brevis	37
Abductor pollicis longus	37
Extensor pollicis brevis	37
Extensor indicis proprius	8
Extensor pollicis longus	5
Extensor communis digitorum	7
Tibialis anticus	7
Extensor hallicus longus	6
Extensor digitorum longus (foot)	3
Extensor carpi radialis (alone) (longus and brevis)	2
Flexor carpi radialis	3
Flexor carpi ulnaris	2
Tendo achillis (gastrocnemius and soleus)	3
Peroneus longus	2
Peroneus brevis	2
Biceps femoris	1
Supraspinatus	1

* Associated muscles are often simultaneously affected.

A comparison of thirty-four cases (of known disability) adequately treated with plaster splinting after a careful study of the muscle groups involved, and twenty-five cases of almost identical muscle distribution and severity of the lesion, shows strikingly the effect of complete immobilization in reducing the total disability, and hastening recovery. The thirty-four cases treated by complete plaster immobilization had an average disability of 11.6 days, and an average observation period of 16.8 days. Twenty-five cases treated mainly by straight board splints, occasionally leaving the thumb free, gave an average of 22.6 days of disability period, and the observation period averaged 27.3 days. If one chooses from both groups the cases in which inadequate immobilization or too short recovery periods of splint rest were followed by diathermy, baking, massage, and strapping, we find that their average disability period becomes 45.1 days —a striking illustration of the value of rest. Surely then, massage, baking and diathermy have no place in the treatment of this lesion.

The time factor of trauma is important in the development of chronic lesions of the locomotor apparatus as well. The trauma consists both in the internal stresses to which our tissues are subject from muscular exertion, and the minor continued external trauma to which every individual is exposed in daily living, be his occupation sedentary, hazardous or active.

"Subdeltoid bursitis" is such a lesion. This disease rarely presents an acutely distended swollen bursa. Fluid cannot be obtained on aspiration in the usual case. Acute serous bacterial or purulent inflammation of the bursa is almost unheard of. In direct traumatic soft tissue lesions of the shoulder, lacking fracture, dislocation, or muscle tendon rupture, one does not find a bursa distended with blood. These phenomena accompanying the common prepatella and olecranon bursitis, traumatic or infected, are lacking.

A. W. Meyer[5] has repeatedly shown that attrition changes due to wear and tear are frequently present in the human shoulder. The tendons attached to the greater tuberosity of the humerus form the floor of the subdeltoid or subacromial bursa. These tendons, most often the supraspinatus tendon, show fraying, splitting longitudinally, and fragmentation and necrosis, frequently to the point of partial or complete rupture of the supraspinatus tendon.

Barr,[6] in an excellent review of pathologic calcification defines dystrophic calcification as the deposit of lime salts in dead or dying tissue. White fibrous, tendinous, and elastic tissue are most often involved. The available evidence indicates that dystrophic calcification is dependent on local conditions, and is not initiated,

although it may be modified by systemic disturbances in calcium metabolism.

Sandstrom and Wahlgren[7] give clear radiologic details correlated with histologic findings from operative material, and in a splendid article show definitely that the dystrophic calcification of so called "subdeltoid bursitis" actually lies in the tendon and peritendon of the muscles attached to the greater tuberosity. From his extensive material, Sandstrom gave this condition the name "peritendinitis calcarea" in an earlier publication. He was able to show that most instances of bursitis calculosa, not only in the shoulder, but in other regions, are really peritendinitis and tendinitis calcarea. The muscle itself may be involved in this process, which is not an ossifying myositis or peritendinitis following acute trauma, but develops gradually over long periods of time. Peritendinitis calcarea is not associated with bacterial inflammation, nor with rheumatic fever or gout, but is due to dystrophic calcification in areas devitalized by the attrition changes described by Meyer. Milch and Green[8] have recently described a series of cases in which the flexor carpi ulnaris tendon was involved.

In the treatment of this lesion in the shoulder, great changes have arisen. In the acute painful "subdeltoid bursitis" we have been able to obtain relief of pain and restoration of active shoulder motion by the simple injection of 15 to 20 c.c. of 1 per cent novocaine into the subdeltoid bursa, followed by gentle manipulation of the shoulder through its full normal range of motion. It is not actually necessary to puncture the calcium bearing deposit and wash out the bursa. From Sandstrom's studies, one can easily state that no irrigation could completely remove the amorphous calcium salts which are present in tendon and peritendinous tissues, and which only rarely break through into the actual bursal cavity. What change does occur cannot be described. Certainly, the disabling symptoms are not dependent on the presence of the calcium alone. Frequently the opposite, apparently normal, symptomless shoulder shows as much or more dystrophic calcification as the ailing one. Following the relief of symptoms the calcium may disappear to roentgenographic visualization in as early as eight days.

Injection of the bursal region with novocaine has supplanted operation since my colleague, Ralph Soto-Hall[9] began the treatment in 1933.

In a series of eighteen cases of acute subdeltoid bursitis, personally treated by the injection method, the average disability was 4.7 days. A number of these patients were able to return to their work the following day. One patient was disabled for twenty-eight days. In several instances, residual soreness, but not acute pain, persisted for from three to five days. Comfort was maintained during this period with the use of a sling, and with diathermy. No such response and quick return to work occurs when treatment consists of abduction splinting, diathermy or roentgenotherapy.

Less spectacular and effective is the injection treatment in those chronic cases with muscle contracture and atrophy (scapulohumeral fibrosis) the result of long standing painful peritendinitis calcarea of the shoulder. The injection may be of help in relieving pain, but usually must be supplemented by balanced arm traction and bed rest, followed by the use of an abduction splint with graduated exercise. Such scapulohumeral fibrosis with painful shoulders may be prevented by early injection treatment.

De Quervain's[10] stenosing tendovaginitis of the extensors of the thumb may be accompanied by gross crepitus, softer in quality than that found in peritendinitis crepitans. The pain on movement may be disabling, but the tenderness and swelling are confined to the synovial sheath.

In six such cases that I have seen, simple conservative treatment was carried out with complete recovery and no disability after initiation of treatment. Only one patient (bilateral involvement in an elderly

typist) gave no previous history of previous injury to the lower radius, or had no projecting gross bony ridges or exostosis from arthritis. Previous fracture of the lower radius had occurred, three months, one year, and two years before development of symptoms. By the wearing of a moulded leather wrist splint, extending beyond the distal joint of the abducted thumb, leaving the fingers free, all were relieved of symptoms, and all symptoms disappeared. The longest course was two months. Each patient continued her occupation (housewife, social worker, typist, and piano player in a night club). De Quervain saw one patient with crepitating peritendinitis later develop the stenosing tendovaginitis called by his name.

True acute traumatic tenosynovitis occurs following direct contusion trauma or puncture wounds. This is not bacterial infection, but extravasation of blood and synovial fluid within the sheaths. Fine crepitus may be heard with a stethoscope over the sheaths on movement, active or passive, of the tendon. Acute pain is unusual; the discomfort is dull and aching in type, with a feeling of fulness. I have encountered nine instances of hemorrhagic effusion into tendon sheaths, either from direct blunt trauma or puncture wounds. In each case the tendon sheath was distinctly outlined by the swelling. Such effusions disappear promptly with simple immobilization, and disability rarely extends beyond a week.

A very rare tendon lesion is the "tenosynovitis fungosa" of the Germans.[11] Two such cases have come under my care. Both were located within the tendon sheath of the extensor communis digitorum. The effusion was not accompanied by local or systemic signs of infection. No direct trauma was associated, although both were in trades involving flexion and extension of the wrist at high speeds for long hours. The fluid aspirated from the sheaths was mucoid, brownish tinged and had a turbid opalescence. Microscopically, one found numerous vacuolated phagocytes and round cells having a hydropic appearance, occasional red blood cells and isolated endothelial cells. No organisms were obtained on smear or culture, and guinea-pig innoculation, as well as culture for acid-fast organisms, was negative.

Since prolonged splinting or strapping did not relieve the condition, and diathermy was of no help, operative exposure was carried out. The synovial sheaths were found boggy and edematous, but with very little congestion and hyperaemia. In each instance, the extensor digitorum communis slip to the index finger and the extensor indicis proprius tendons showed ragged, brownish fungus granulations near their disappearance beneath the dorsal carpal ligament. The tendons involved exhibited gross fraying and partial ruptures. The granulations were excised, the external wall of the tendon sheath cut away, and the dorsal carpal ligament divided over about one-third of its width and the skin and subcutaneous tissues carefully sutured. The wrist and fingers were held immobilized in plaster for two weeks. Paraffin blocked microscopic sections showed no tuberculosis in the chronic non-specific granulation tissue and no evidence of lues. The blood Wassermann reaction was negative in both cases. The first patient had been treated with potassium iodide for three weeks before operation with no improvement.

We are still ignorant of the true cause of this pathologic change in the tendons. Both patients were able to return to their work within eight weeks and have had no recurrence after almost two years' time.

Finally, we have the group of occupational cramps and spasms allied to writer's cramp, found in this day in milkers, seamstresses, and the like, where fine rapid motions of the fingers, usually necessitating exertion of considerable force, are carried out. These seem to be mainly dependent upon exhaustion phenomena in the interosseous and lumbricalis muscles, occasionally in the muscles of the thenar and hypothenar eminences.

DISCUSSION

Lee[12] remarked in 1906, that "it is a striking principle of biology that activity of living substance tends to inhibit its further activity. Carried to a moderate degree, this inhibition leads to self preservation of the living substance, to an extreme degree to its self destruction. Fatigue is a comprehensive term, and lying on the border zone where the physiologic and pathologic meet, it reaches far into both and obscures the division lines between them." Palmer,[13] in an interesting discussion on the mechanics of injury, refers to Baetzner who limits Roux' law of adaptation through function. Baetzner[14] feels that functional adaptation of an organ or tissue occurs only within certain limits, beyond which, instead of functional adaptation, is found lower resistance and pathologically changed or vulnerable tissue. He named such changes a "pathology of function."

It is shown that certain instances of disease cited in this paper, particularly crepitating peritendinitis and peritendinitis calcarea, are acute and chronic stages of pathology of function, not necessarily occupational in nature, and that perhaps the future will show more clearly the relation of other diseases of the locomotor skeletal apparatus to similar long continued forces of stress, compression and strain.

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DISEASES OF THE PERIPHERAL ARTERIES AND THEIR REACTION TO TRAUMA

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INSIDIOUS onset, cause unknown, affecting adults of all ages but most frequently those in the prime of life—and mostly men—diseases of the peripheral arteries, of whatever nature, present a problem interesting, perplexing and by no means solved. Especially significant and unfortunate is the onset. If only the disease began with the severe pain that is such a prominent feature later on, or with an intractable ulcer or with an inability to walk!

It does not do that, of course, and in the nature of things could not, because the pain, the ulcer, and the inability to walk, coming late as they do, indicate profound vascular disturbance—which isn't the case early in these affections. In the beginning there may be only some vascular irritability, slight spasms of terminal vessels giving rise to more or less fleeting color changes of fingers or toes, or perhaps an occasional area of phlebitis in the superficial veins, migrating phlebitis, so-called, more characteristically noted in thrombo-angiitis obliterans or Buerger's disease. Excessive sweating of the lower limbs and hands is an early sign, too, an index of sympathetic nervous system derangement.

But it is all insidious. There is nothing definite about it. And if one were to cut down on the blood vessels of the limbs so affected they would show little if anything to the naked eye and not much more microscopically. Only later, when real cramps in the calf muscles come and when the patient can walk but a few blocks on account of those cramps or when he begins to have the tell-tale red foot and pain in the toes, more especially the big toe, worse at night and causing loss of sleep, would the blood vessels show real changes. Then, of course, the more terminal ones would be occluded by thrombi, or obliterated by sclerosis or some other inflammatory process, while even the major ones might be profoundly affected—even to the point of obliteration.

About the only thing of possible causal nature patients suffering from peripheral vascular diseases have in common is tobacco. Practically all of them are great cigarette smokers. Curiously enough, syphilis is of little importance—the endarteritides of this origin being fewest in number. In fact, those who suffer from maladies of the peripheral blood vessels usually have lived exemplary lives, their histories being singularly uninteresting. That is a strange thing. Most of them are married, have children, haven't drunk to excess or done anything else to excess—except smoke, and once in a great while they haven't even done that.

It is generally felt, and I think proved, through years of observation on hundreds of cases, that, even if it is not the actual cause of vascular disease, tobacco is at least harmful to the blood vessels of certain people and makes a bad situation worse. It seems to irritate the peripheral vessels, causing at first mild spasm and later major ones, finally ending in their complete occlusion. There is dispute as to whether the trouble is due to the nicotine content or some other element in the tobacco. But that there is a disturbing element there is little doubt. Why it affects some people, though, and not others, is a question that has yet to be answered. One can only rationalize. Certain people can consume great quantities of alcoholic beverages, and in short order, without being affected, while others have only to take one or two drinks to be much affected, even intoxi-

cated. Why could it not be the same with regard to tobacco—poison even in moderate quantities to the minority and affecting their peripheral vessels chiefly and profoundly, while being innocuous to the great majority, even in great quantity.

Diabetes, of course, is another contributing cause of peripheral vascular disease. Regardless of age and the thoroughness of treatment, the blood vessels of the diabetic always show definite arteriosclerotic changes as a result of which their limbs are vulnerable. Of all people, the diabetic should avoid trauma. Not once but many times have I seen a tight shoe act as the starting point of an intractable ulcer. And upon a number of occasions abrasions around the toe nails or visits to the chiropodist have been followed by tragic consequences. Women seem to be more affected by diabetic gangrene, in my experience, than men, but more of them have diabetes than men. Therapeutically, they do about the same.

Those suffering from thrombo-angiitis obliterans or Buerger's disease are mostly men. Only rarely are females thus affected which, by the way, is an argument in favor of the tobacco contributing cause theory. Women have not been great smokers. They are certainly smoking now though, and it will be interesting to note whether more of them will come down with the disease in future years.

However the case may be, most of those who have thrombo-angiitis obliterans are men between the ages of 20 and 60, the majority between 35 and 45. My youngest patient was 16 when his disease began—a boy of Russian-Jewish descent, who eventually died at 42, having lost all four extremities. The disease was at first supposed to affect exclusively Russian Jews or Jews of Russian descent, but we know now that it is by no means confined to the one race. Within the week I saw in consultation an aggravated case in a young army officer of pure American stock. He was only 26, and said his father had had the same malady before him; in his father's case, the thing began when he was 54 and eventuated in the loss of both legs. Beginning at that age, it may have been an arteriosclerosis or obliterating endarteritis that the father suffered from. I have not noticed any hereditary tendency in Buerger's disease, though I have often seen vascular imbalance, varicose veins, sclerosis, and mild Raynaud's disease in families.

The Buerger's disease patients frequently have somewhat of a low blood pressure and numbers of them are thin, underweight, and not too robust—not the majority, but a considerable number. They practically all are diagnosed rather late. Physicians too often content themselves with treating these individuals for the minor grades of varicose veins and flat feet that they, and lots of others, show, rather than search for less apparent but more important underlying causes of disability. Only after elastic stockings and arch supports have given no relief is suspicion of the true condition aroused. Meantime, much valuable time has been lost. Nor is this all. Patients suffering from peripheral vascular disease most certainly ought to be warned against trauma. How can they if the nature of their condition is not known?

A case in point was that of a man 55 years old who first noticed an abrasion above the heel. He reported to his company doctor who treated it in the usual way, but a small ulcer developed which simply refused to heal. Inasmuch as there was little pain the man was permitted to continue working, with the result that the ulcer continued to enlarge, the tissues about it became infected and he was sent into the hospital. There the true nature of the trouble was first discovered. The man had generalized arteriosclerosis—most marked in the affected leg where no pulses whatsoever were to be felt. In spite of the most careful treatment the infection spread and gangrene ensued. Amputation failed to save him.

Another case similar in character was that of a middle-aged man upon the dorsum of whose foot a piece of metal accidentally

fell. The man was working in a foundry at the time, and he, too, was sent to the company doctor. After weeks and weeks of unsuccessful treatment the patient was hospitalized, but by this time there was a spreading gangrene which finally ended in death. An unrecognized Buerger's disease was the cause.

Pain and inability to walk, so-called intermittent claudication, are the two outstanding features of peripheral vascular disease pretty much regardless of the type. In other words, whether the diagnosis in a given case is Raynaud's disease endarteritis obliterans, arteriosclerosis, Buerger's disease, or anything else, sooner or later there is pain and difficulty in walking.

Later the patient begins to lose sleep, his appetite fails, and a general debility of greater or lesser proportions supervenes. Indeed, these maladies are general in character, the signs in the extremities being but local manifestations. One rarely finds arteriosclerosis confined to one leg or arm any more than one finds obliterating endarteritis located in only one extremity. The whole body is involved, with the one limb being the most affected. It is not at all infrequent for patients suffering from thrombo-angiitis obliterans or Raynaud's disease to have a cerebral accident and upon occasion I have seen a mesenteric thrombosis. One of my patients, a young man of 28, came down with such a catastrophe and only recovered after a resection of six feet of his small bowel. Mention is made of this because of its therapeutic importance. Naturally one cannot very well prevent a cerebral accident or a mesenteric thrombosis, but if he recognizes the fact that all peripheral vascular diseases are general in character and handles the patient accordingly by rest and such other measures as may be calculated to relieve the general condition, the chances of their occurrence are at least minimized. More than that, general supportive measures do much to alleviate the local manifestations. Rest in bed and a bit of digitalis help a weakened heart and bolster a failing circulation—as does relief of pain which permits sleep.

It follows, therefore, that the earlier one recognizes peripheral vascular disease the better the prognosis. All patients, regardless of the type of disease, should give up entirely and for life the use of tobacco. They should have much rest—in bed if the condition is at all aggravated. Ulcers and infections should be treated with extreme conservatism—wet dressings of mild antiseptics, with frequent bathings to prevent the collection of secretions. Devitalized tissue had best be permitted to slough out, since in the presence of an impaired circulation, operation only too often aggravates the condition.

If there is no infection or but little, the Pavaex may be used, or diathermy. Ultraviolet light is at times helpful. Gangrenous toes or fingers had best be permitted to mummify and drop off. This process takes weeks, but is painless, and nature frequently does a successful amputation where man utterly fails.

Personally, I do not like the various exercises usually prescribed, preferring mild massage. And I let my patients do only such walking as is absolutely necessary—only up to a point at which a cramp *may* come on. They are warned against actually getting cramp. Gradually, as their collateral circulation improves, they come to walk better and farther. I frequently give them 250 c.c. of 3 per cent hypertonic salt solution intravenously three times a week in preference to other forms of therapy and with better results. As they improve they get less salt and at more infrequent periods. Samuels* is a great exponent of this treatment which, in his hands and in others, including my own, has given excellent results. Patients need not be hospitalized for the treatment. I have used it in all forms of peripheral vascular diseases, but it seems to help sclerosis and thrombo-angiitis obliterans most.

* Samuels, S. S. The Diagnosis and Treatment of Diseases of the Peripheral Arteries. Oxford University Press, 1936.

In conclusion, then, it may be said that peripheral vascular disease is noted most frequently in men who are in the prime of life. The onset is insidious with difficulty in walking because of pain in the calf muscles gradually becoming prominent. In view of the fact that early diagnosis is so important, the peripheral circulation, more especially the arterial, should be checked in every individual who complains of the slightest difficulty in walking. Once a diagnosis of peripheral vascular disease is made especial emphasis should be laid on the avoidance of trauma. Rest and the therapeutic aids suggested give very satisfactory results.

LATERAL condyle fractures in children may often be successfully treated by closed manipulation and retention in flexion. Open reduction may be necessary and should be practiced in all cases in which the fragment is so rotated that its articular face is directed medially.

PERIPHERAL NERVE INJURIES AND FRACTURES*

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IN the older reports of cases of peripheral nerve injuries, it was usually stated that they very rarely occurred as complications of fractures. No doubt that was true before the days of the high speed automobile or of so many industrial accidents. Nowadays, however, where greater force is involved in accidents, peripheral nerve injuries are becoming increasingly common. For that reason anyone who undertakes to practice traumatic surgery must be constantly aware of the possibility of injuries to peripheral nerves as well as to the blood vessels or other soft tissues. Moreover, there is the additional responsibility of determining whether the injury to the nerve is a temporary edema or if there has been a permanent break in continuity, since subsequent treatment depends upon this decision.

It is not within the scope of this paper to discuss fractures of the skull or fractures of the spine—two conditions which are frequently associated with evidence of peripheral nerve damage. Also, no cases of birth injury to the brachial plexus (Erb's palsy) will be included. The main concern here is a typical group of patients with different types of injuries to peripheral nerves.

As is well known, the commonest nerve injuries which accompany fractures involve the radial, ulnar, and peroneal nerves because they are situated in vulnerable locations. This is easy to understand when we realize that the radial nerve is directly in contact with the middle third of the shaft of the humerus as it winds around the musculospiral groove. The ulnar nerve likewise is subcutaneous at the back of the elbow and in contact with the medial epicondyle of the humerus. And in the leg the common peroneal nerve is subcutaneous in the region of the head of the fibula and in immediate contact with the bone. While all the major peripheral nerves are subject to injury, these three nerve trunks are the ones most exposed to lacerations secondary to fracture.

A simple contusion of the nerve trunk at the time of injury tends to be overcome rapidly. In a few weeks, after the acute edema subsides, motor and sensory functions gradually return. The last stages of recovery may take months or may never occur, but if any recovery is taking place at all, it is proof that the nerve is not severed. If, on the other hand, there is no such return of function, and complete motor and sensory loss persist unchanged, it is likely that the nerve has been lacerated extensively, bound by scar tissue or callus, or severed at the time of the accident. Moreover, if recovery does not begin to manifest itself within three or four weeks, the affected nerve should be explored in order to correct or remove the cause of the disability.

The injured nerve must be examined for evidence of laceration or neuroma formation. Such tissue is removed by sharp dissection until normal nerve is exposed at each side of the laceration. The nerve ends are then brought together (without tension or rotation) and the epineurium sutured with fine silk or linen. Catgut is unsuited to this purpose because it is not sufficiently strong to protect the spliced joint and there is often a chemical reaction about it that is injurious to nerve growth.

Nerve grafts or transplants are only occasionally successful and of dubious value. If there is a large gap in the nerve, nearby joints can be flexed to permit the

* Read before the Texas Surgical Society, Houston, April 4, 1938.

nerve ends to come together or some nerves (as the radial or median) can be rerouted in the extremity. In other cases, it is possible to place the proximal nerve end in the belly of the paralyzed muscle. As the nerve cells grow outward, motor endings are formed which take up the function of the damaged nerve ends.

In this study there were 25 patients with injuries to the various peripheral nerves, ranging from simple contusions to complete ruptures. Not all of them accompanied fractures of the long bones although trauma was the precipitating factor in each case.

Nerve Involved	No. of Cases
Common peroneal	8
Radial	5
Ulnar	5
Sciatic	2
Median	2
Brachial plexus trunks	2
Intercostal	1
Total	25

INJURIES TO THE COMMON PERONEAL (EXTERNAL POPLITEAL) NERVE

The common peroneal nerve branches from the sciatic trunk, curves laterally in the popliteal space and forward around the neck of the fibula. It is superficial as it crosses the fibula and can be palpated beneath the skin. It supplies motor branches to the muscles which dorsiflex the ankle and toes (anterior tibial, extensor digitorum longus) and which evert the foot (peroneus longus and brevis). Cutaneous sensory branches supply the lateral side of the leg and foot. Therefore, when this nerve is severed there is loss of extensor power in the ankle and toes and loss of eversion of the foot as well as anesthesia over the lateral margin of the leg.

The most serious injury among these cases resulted from a fracture-dislocation of the knee in which the peroneal nerve was lacerated extensively in the popliteal space. Another case of peroneal nerve laceration resulted from a fracture of the shaft of the femur which had been manipulated several times. This injury had occurred a number of years before so that nerve suture was out of the question. Therefore, arthrodesis of the ankle was advised. Two of the patients had developed foot drop from carelessly applied adhesive skin traction on the leg which caused undue pressure over the head of the fibula. One patient with an old fracture of the head of the fibula had a persistent foot drop.

In manipulating fractures of the shaft of the femur or of the upper end of the fibula, it must be borne in mind that nerve trunks can be injured if the fragments are moved about carelessly. Moreover, the region about the head of the fibula should always be protected against undue pressure when skin traction or casts are applied to the leg. This region likewise should be carefully protected during operations or at any time when an anesthetized patient is on his side.

If the symptoms which follow the injury to the peroneal nerve suggest a laceration or if the disability shows no evidence of subsiding within three weeks, the nerve should be explored and repaired. The chance of eventual complete cure is much better if the repair is done early. Eighteen months or more after the accident it is generally futile to attempt to suture the nerve, and in such cases an arthrodesis of the ankle or tendon transplant at the ankle is the treatment of choice. Sometimes the superficial peroneal nerve alone is injured, which causes loss of power of eversion of the foot, while the deep peroneal branch may be intact in its supply to the anterior tibial muscle. If so, the tendon of the anterior tibial muscle can be transplanted to the cuboid bone on the lateral border of the tarsus to overcome the inversion deformity.

CASE REPORTS

Case 1. O. T., a 19 year old male had had a fracture-dislocation of the left knee three months before, followed by complete foot drop, inversion deformity, and numbness down the lateral side of the leg. The peroneal nerve was explored in the popliteal space and a lacerated area 1 inch long removed. The nerve was

sutured by end-to-end anastomosis. A cast was applied with the knee in acute flexion.

Case II. A. S., a male of 16, had had the skin over the lateral aspect of his left thigh and leg badly lacerated in a bus accident ten years before. There had been a persistent inversion deformity of the foot due to paralysis of the peroneal muscles (supplied by the superficial peroneal nerve). The anterior tibial muscle (supplied by the deep branch of the peroneal nerve) was intact. The anterior tibial tendon was transferred to the cuboid bone which caused marked improvement in gait and function of the foot. (Fig. 1.)

Case III. L. O. B., male, 22, reported an old fracture of the head of the fibula with inversion deformity of the foot due to paralysis of the peroneal muscles. The anterior tibial muscle which was intact was transplanted to the cuboid bone. The gait was much improved and there was less tendency to stumble or fall.

Case IV. T. D., a girl of 7, fractured the right femur ten months before. She had been treated by vertical traction on both legs with adhesive Buck's extension. The skin on the right leg became macerated and there was a foot drop deformity afterward. The peroneal nerve was undoubtedly lacerated and should be explored. She is to wear an ankle-catch brace meanwhile.

Case V. W. M., male, 29, fractured the head of the right fibula fifteen years ago. He has had persistent foot drop and inversion deformity with numbness on the lateral side of the foot. He was advised to have a bone block operation of the right ankle to correct the "steppage gait."

Case VI. M. J., a boy 10 years old, had a fracture of the middle third of the right femur treated by adhesive skin traction and Thomas splint. He has had a foot drop for six months with only slight recovery. If there is no more improvement in the next three months, the nerve should be explored. He is to wear an ankle-catch brace meanwhile.

Case VII. D. B., a 44 year old man, had persistent foot drop on the right after a kidney operation, probably due to direct pressure on the head of the fibula while the patient was on the operating table. He was given baking and massage to the leg muscles and an ankle-catch brace while awaiting signs of recovery.

Case VIII. A. S., female, 23, had fractured the left femur fifteen years before. The leg was manipulated under anesthetic several times and there has been foot drop since. She was advised to have a stabilization of the ankle.

INJURIES TO THE RADIAL (MUSCULOSPIRAL) NERVE

Even though it is not superficial, the radial nerve is very susceptible to injury. Dislocation of the shoulder, fracture of the humerus or a carelessly applied tourniquet about the arm may cause pressure neuritis or laceration of the trunk of this nerve. Drunkards or patients under anesthetic sometimes suffer radial injuries if there is too much pressure on the posterior surfaces of the arms. "Drunkard's palsy" often follows a night's sleep in a chair or across a table.

It is obvious that radial nerve injuries can be forestalled if manipulations of the shoulder or of a fractured humerus are performed carefully and with a minimum of force. Moreover, when patients are under an anesthetic lying on a hard table, the arms should be padded so that there is not undue pressure against them.

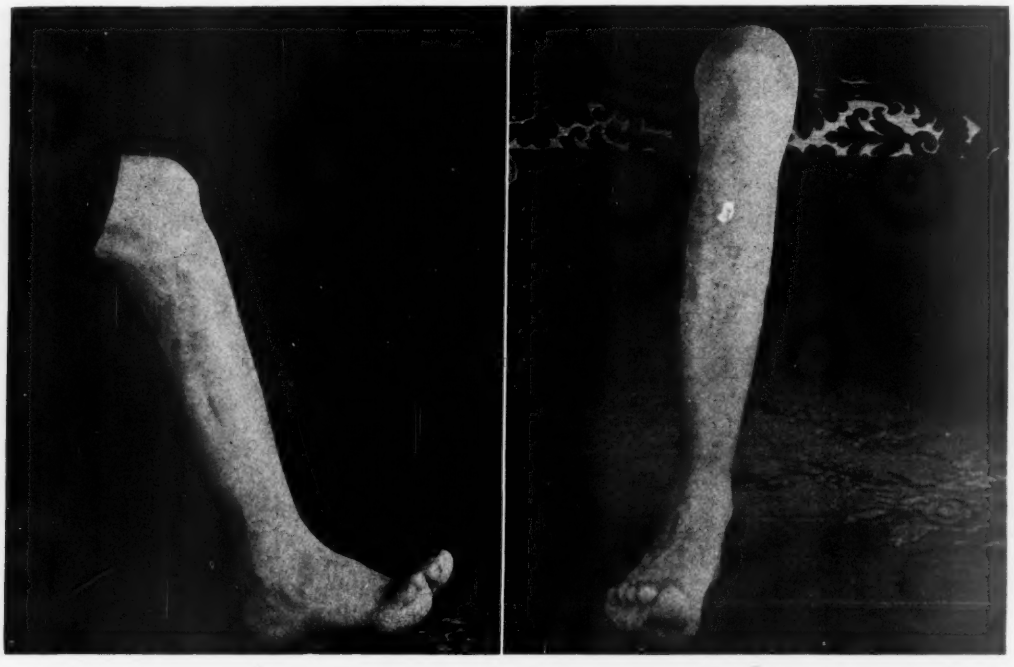
Injury to the radial nerve is manifested by loss of extension power of the thumb, fingers, and wrist ("wrist drop"). If the injury is high in the arm, there will also be loss of extension of the elbow since the triceps muscle is supplied by this nerve. Sensory loss is over the dorsum of the thumb and index finger and over the web between them. These evidences of sensory loss, however, are very variable because other sensory nerves overlap in the region supplied by the radial nerve.

Suture of this nerve is highly successful since it is a predominately motor nerve and is the largest branch of the brachial plexus. Moreover, if a portion of the nerve needs to be removed, acute flexion of the elbow will provide enough slack to permit the ends to be brought together without undue tension.

CASE REPORTS

Case IX. I. M., a man 40 years old, suffered from an old osteomyelitis of the humerus with

sinus formation. The radial nerve was bound in a scar on the lateral side of the arm, producing a wrist drop and anesthesia over the dorsum of the thumb. The nerve was dissected free and transplanted away from the scar. Gradual complete recovery ensued.



A B

FIG. 1. Case II. Laceration of the lateral aspect of the leg ten years before. Inversion deformity of the foot due to paralysis of the peroneal muscles. A, old laceration of the lateral aspect of the leg. B, anterior view showing ability to evert the foot after anterior tibial transplant.

CASE X. A. M., a 19 year old female, fractured the left humerus three months before. It had been manipulated several times, and there was a complete wrist drop afterward with no sign of recovery. The nerve was explored and found to be bound in dense scar tissue on the posterior aspect of the humerus. A fibrous segment of the nerve was removed and the ends sutured. Complete recovery ensued except for failure to recover abduction power in the thumb. (Fig. 2.)

CASE XI. J. M. L., male, 32, gave a history of gunshot fracture of the humerus one year before. There was non-union of the fracture and wrist drop after several months. A bone graft was placed in the defect in the humerus. Then the nerve was explored, the neuroma removed, and the nerve ends sutured. The patient recovered extensor power in the wrist and almost complete extension of the fingers. However, the thumb could not be abducted. (Fig. 3.)

CASE XII. M. D. E., a 17 year old girl, had been stabbed in the forearm two years before. The old adherent scar on the palmar aspect of the right forearm had produced numbness and pain over the thumb and index finger. The scar was dissected out and the radial nerve trunk freed. The patient recovered sensation on the dorsum of the index finger but partial anesthesia of the thumb persisted. The pain was relieved.

CASE XIII. C. F. L., a male of 54, had an old fracture of the lower third of the shaft of the radius with non-union of the bone and numbness over the thumb and index finger. The bone was tied with kangaroo tendon. Much of the numbness persisted although "the tight feeling" was relieved.

INJURIES TO THE ULNAR NERVE

The ulnar nerve passes behind the medial epicondyle of the humerus and into the forearm. It supplies the intrinsic muscles of the hand and sensation over the little finger and half the ring finger. Consequently, if this nerve is damaged or severed, atrophy develops in the interspaces and hypothenar eminence of the hand. The fingers become hyperextended (due to the paralysis of the short flexors in the hand) and the result is the typical

"claw hand." Numbness is produced over the ulnar border of the palm of the hand, the little finger, and half of the ring finger. Here again there is much variability in sensory loss due to overlap from the other nerves which supply the hand.

Fig. 2. Case x. Fracture of the left humerus three months before with complete wrist drop afterward. The radial nerve was found to be densely adherent to the posterior aspect of the humerus. Damaged portion removed and nerve ends sutured. Picture taken one year later shows ability to extend the wrist and fingers. There is still inability to hyperabduct the thumb.

The "claw hand" of ulnar nerve injury must be distinguished from the similar type of deformity produced by Volkmann's ischemic contracture. This latter condition sometimes is caused by injuries about the elbow that interfere with the blood supply to the flexor muscles of the forearm. The distinct features of Volkmann's paralysis which are not present in ulnar injury cases are: No sensory changes in the hand which follow ulnar distribution, induration of the flexor muscles of the forearm, and generalized atrophy of the hand due to inactivity.

"Tardy ulnar nerve paralysis" is a condition in which symptoms develop ten to thirty years after a fracture of the elbow in childhood. Apparently as the arm grows, a valgus deformity of the elbow develops and the ulnar nerve becomes stretched unduly. This produces tingling pain along the ulnar margin of the hand and atrophy of the intrinsic muscles.

Fig. 3. Case xi. Gunshot fracture of the right humerus one year before with non-union of the shaft of the bone and wrist drop deformity. Bone graft applied to the humerus. Three weeks later, neuroma removed from the radial nerve. Photograph taken one year later shows ability to extend the wrist and fingers and beginning abduction power in the thumb. (Complete abduction of the thumb has since been recovered.)

If the ulnar nerve is lacerated, it should be sutured after the damaged portion is removed. On the other hand, where there is too much tension on the nerve as in "tardy ulnar palsy" or if pressure symptoms develop, the nerve should be transplanted anteriorly. In this procedure the nerve is lifted from its groove behind the humerus and moved forward in the flexor group of muscles. This usually relieves the pain symptoms. The recovery of the intrinsic muscles in the hand, however, is slow and may take twelve to eighteen months to fill out to the normal contour.

CASE REPORTS

Case xiv. N. A., a female of 36, fractured her elbow twenty-two years ago. Recently she has developed atrophy of the muscles of the hand and pain in the fourth and fifth fingers. The ulnar nerve was transplanted anteriorly and the symptoms disappeared.

Case xv. S. R. A., a girl of 7, had a fracture of the shaft of the ulna four weeks previously. While the arm was in a cast, she developed

clawing of the hand. The ulnar nerve was explored in the middle of the forearm and found to be densely adherent to the healing fracture.

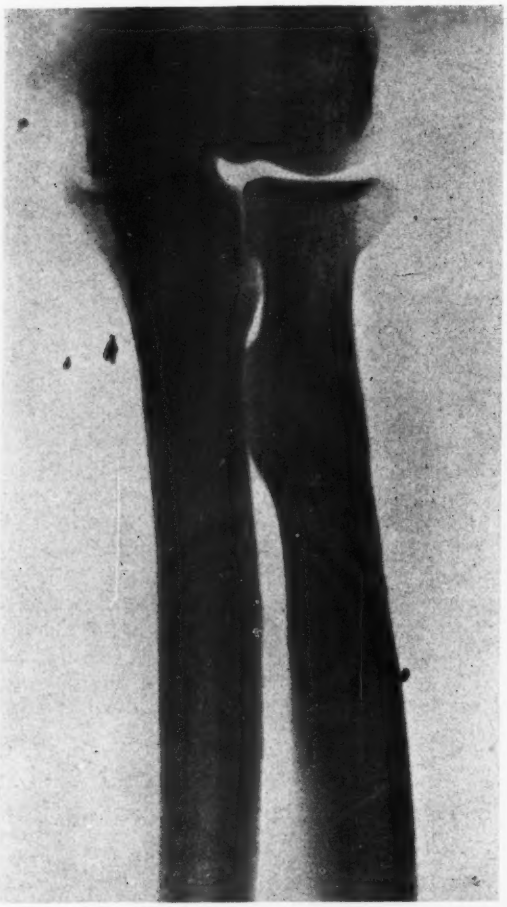


Fig. 4. Case xviii. Oil well explosion six months before caused many puncture wounds over the arms and the body. Photograph shows *x*-ray of the right elbow with metal fragments along the course of the ulnar nerve. Because of atrophy of the muscles of the hand, the ulnar nerve was transplanted anteriorly to a more vascular area.

The nerve was freed from the scar tissue and callus and moved into muscle tissue.

Case xvi. M. H., female, 14, fractured the internal condyle of the right humerus three years before. The condyle had never united with the shaft of the bone. Pain had developed in the little finger in the past year. The old ununited fragment of the bone was removed and the nerve symptoms rapidly subsided.

Case xvii. J. S., a man of 29, fractured the right olecranon eighteen months before. In recent months atrophy of the muscles of the hand was noted. The nerve was transplanted anteriorly and undue tension was relieved.

Case xviii. A. E. H., male, 46, had been in an oil well explosion six months before which had caused many puncture wounds over the arms and torso from tiny metal scraps. There were two such wounds on the posterior aspect

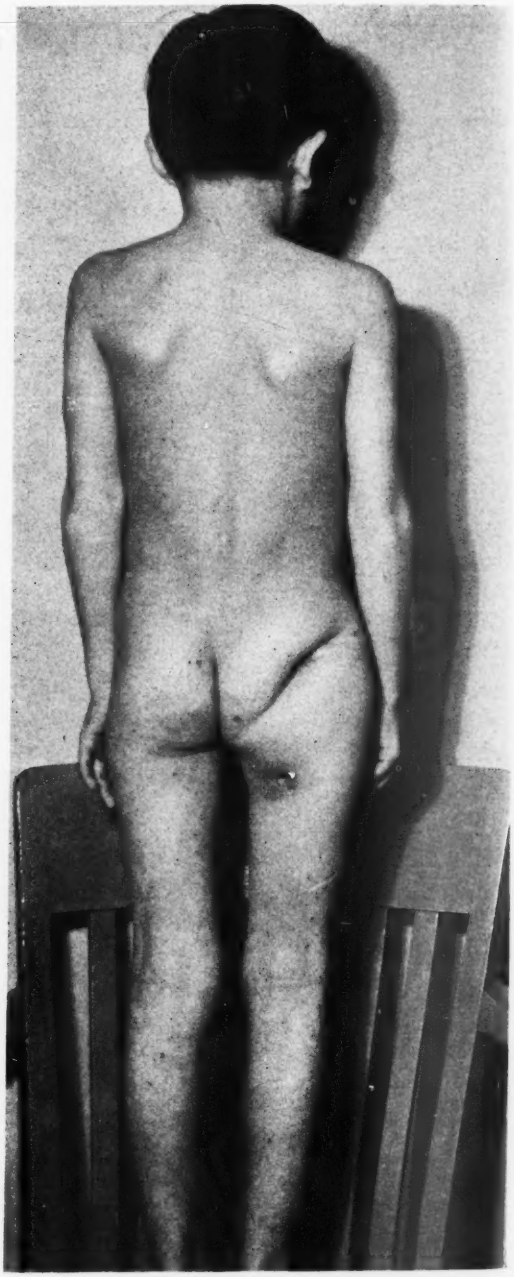


Fig. 5. Case xix. Three years before the patient fell down stairs and lacerated his right buttock on a broken bottle. A dense scar formed in this region. Recently there has been much pain and limp on walking. Photograph shows scar of the right buttock before operation. At operation the scar was found to be bound to the sciatic nerve trunk. Excision of the scar relieved the symptoms.

of the right elbow. Recently he had developed atrophy of the intrinsic muscles of the hand. The nerve was transplanted anteriorly away from dense scar tissue. (Fig. 4.)

INJURIES TO THE SCIATIC NERVE

Dislocation of the hip or fracture of the pelvis may produce injury to the sciatic nerve at the point where it leaves the pelvis. Such nerve injuries are usually manifested by burning pain in the foot or loss of sensation over the foot. Moreover, the motor symptoms are usually those of peroneal nerve injury (see above). In one of these cases there had been a direct blow over the sciatic trunk and in the other there was a deep dense scar of the buttock which surrounded the sciatic nerve.

The proximal axons of the sciatic nerve grow more slowly than in other peripheral nerves so that one should wait at least six months after a suspected sciatic injury to see if spontaneous recovery will take place. Moreover, if the nerve is sutured, return of function is slower than in other nerves because the axons have farther to grow before recovery symptoms are apparent. When we recall that axons grow outward at the rate of approximately an inch a month, it is easy to see why recovery of the sciatic nerve would be slower than in a shorter nerve.

CASE REPORTS

Case xix. J. C., 8 year old boy, three years before fell down stairs and landed on a broken bottle. There was a deep laceration of the right buttock. The wound healed but there had been a marked limp and pain on walking since. The dense adherent scar was dissected from around the sciatic nerve trunk and the muscles brought together over the nerve. (Fig. 5.)

Case xx. L. C., female, 40, fell on a slippery floor and sat down heavily. A large hematoma formed about the left sciatic nerve trunk and produced severe pain down the posterior aspect of the thigh and leg. Pain gradually subsided as the swelling disappeared.

INJURIES TO THE MEDIAN NERVE

The median nerve passes down the middle of the palmar aspect of the forearm, across the wrist and into the hand. In the forearm it supplies the palmaris longus and superficial flexor muscles of the fingers, while in the hand it sends branches to the intrinsic muscles of the thumb. The sensory area of this nerve is the palmar surface of the thumb, index and middle fingers.

Fig. 6. Case xxii. The patient fell from an oil rig two months before and dislocated the right carpal semilunar (lunate) bone. Recently he had noticed pain and numbness in the palm of the hand. The semilunar (lunate) bone was removed. Photograph shows *x*-ray of the wrist on first examination with anterior dislocation of the semilunar (lunate) bone.

When the median nerve is injured or severed, there is atrophy of the thenar

eminence which causes the thumb to fall in the plane of the palm of the hand ("ape hand"). There is also weak flexor power in the index and middle fingers so that the patient is unable to make a fist or to clasp hands normally. In addition he cannot oppose the thumb and little finger. The numbness due to sensory loss is over the palmar surface of the thumb, index, and middle fingers.

The median nerve is prone to injury by stabs or knife wounds about the wrist because of its superficial position. Anterior dislocations of the carpal bones (especially the navicular or semilunar) often cause pressure neuritis of the median nerve. In this connection Abbot has shown that if the wrist is flexed too acutely in treating Colles' fractures, there is likely to be an undue pressure on the median nerve at the proximal margin of the transverse carpal ligament. Since this nerve carries most of the sympathetic nerve supply to the hand, pressure on it may cause the characteristic trophic changes in the hand commonly seen after wrist fractures.

Suture of the median nerve is only partially successful and recovery is slower than is the case with other nerves. Moreover, there is a strong tendency to persistent pain in the hand after such an operation. Weir Mitchell recognized this fact during the Civil War and it was in describing it that he devised the term "causalgia."

CASE REPORTS

Case xxi. W. H., male, 28, attempted suicide by slashing the right wrist. There was immediate anesthesia over the radial side of the palm of the hand and inability to make a fist. The nerve was sutured, and some of the muscular weakness overcome. Nevertheless, pain persisted for many months.

Case xxii. L. R., male, 39, fell from an oil rig two months before and dislocated the right carpal semilunar bone anteriorly. He had much pain and numbness in the palm of the hand. The semilunar bone was removed and the symptoms gradually disappeared. (Fig. 6.)

INJURIES TO THE BRACHIAL PLEXUS

Since there are so many nerves involved, no distinctive symptoms are present after brachial plexus injuries. The sensory and motor signs in the forearm depend on the degree of injury, the location (in the upper or lower nerve roots), and whether there is great hemorrhage or not.

The pain or paralysis generally follows the pattern of one or more of the peripheral nerves of the upper extremity. Frequently severe pain is the predominant symptom.

In most cases the patients are more comfortable with the arm on an aeroplane splint since this tends to relieve the drag on the brachial plexus. It is seldom advisable to explore the nerve roots because the brachial trunks are usually injured where they leave the spinal cord or in the intervertebral foramina. In either case they are relatively inaccessible for surgery. Support to the shoulder and arm, bake and massage, and sedatives therefore constitute the preferable treatment.

CASE REPORTS

Case xxiii. J. A., a man of 46, was in an open roadster when it turned over and he was thrown out of the car. There was immediate left deltoid paralysis and marked weakness of the hand. He used an aeroplane splint for ten months while receiving physiotherapy. Complete recovery followed.

Case xxiv. P. G., male, 39, was in a car which ran into a ditch. The patient was thrown out on his right shoulder. There was weakness of the right deltoid, anesthesia of the thumb and index fingers, and severe pain in the right shoulder. Recovery was spontaneous except for tingling in the thumb and index finger.

INJURIES TO INTERCOSTAL NERVES

The intercostal nerves pass around the thorax along the inferior margins of the ribs. In fractures of the ribs they are rarely injured because there is seldom gross displacement of fragments. On the other hand, they are especially liable to injury after

fractures of the head or neck of the rib, at the point where it joins the spine. If, after such a fracture, intercostal neuralgia is sufficiently severe, it may require section of the nerve or injection with alcohol. The treatment of acute cases is the treatment of any severe injury to the chest (rest on a stiff bed, immobilization of the chest, etc.).

CASE REPORT

Case xxv. L. W., 35, fell off a ladder four months before and landed on his left side. There were no demonstrable fractures, but he had had persistent pain along the second rib which was not relieved by strapping the chest. He was advised to have an alcohol injection of the nerve.

SUMMARY

1. The common peroneal, radial, and ulnar nerves are quite susceptible to injury in fractures of the long bones.

2. The preliminary examination of a fracture case should include search for any evidence of peripheral nerve damage.

3. In the manipulation of fractures and application of retentive apparatus, the location of the major nerve trunks should be borne in mind so that they will not be injured needlessly.

4. In a series of twenty-five cases of peripheral nerve injury, the distribution was found to be as follows:

	Per Cent
Common peroneal nerve	32
Radial nerve	20
Ulnar nerve	20
Sciatic nerve	8
Median nerve	8
Brachial plexus	8
Intercostal nerve	4

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TREATMENT OF INDUSTRIAL BURNS

WITH REPORT OF 350 CASES

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THE treatment of industrial burns properly begins with prevention. A surgeon charged with the responsibility of treating injured employees should adequately instruct both the safety and first aid attendants in the prevention and early care of burns. This is imperative where the burn hazard is great.

The method used in treating the industrial burns in this series has been moderately successful, and is given in detail for what it may be worth. The first aid attendants are instructed that when a burn occurs they should first put out the fire and remove all the patient's clothing. This is important, as it is disconcerting to receive a patient at the hospital whose clothing is smoldering or still contains chemicals. If the burn is from chemicals the area is washed copiously with water. If the burn is electrical, and it is necessary, the patient is given artificial respiration using oxygen and carbon dioxide until he regains consciousness. After this the treatment is the same for all burns. The patient is given an injection of morphine, the burned area is covered with several layers of sterile gauze and then bandaged. The entire dressing is saturated with 5 per cent solution of tannic acid freshly prepared. This solution is kept readily available by keeping weighed amounts of tannic acid in double paper bags tied about the necks of large bottles containing a measured amount of sterile, distilled water. The corner of the bag is torn and the powdered acid dumped into the bottle at the time of use. Large dressings are autoclaved and kept available. After the dressing is saturated, the patient is wrapped in blankets, given as much water as he will drink and sent to the hospital.

When a burn has occurred, it should be regarded as a serious injury, and the patient should be treated accordingly. Since Davidson[27] introduced tannic acid for the treatment of burns there has been a marked improvement in the mortality and morbidity,[36] but it should be remembered that tannic acid, or any modification, is only a local application, which treats the local injury. This local injury, however, has far reaching results which produce symptoms varying with the age of the patient, the area burned, the depth, the extent, and the agent causing the burn. Mitchiner[52] has shown that the mortality from scalds is only half that of burns, where the treatment used is the same.

I wish here to present a modification of the tannic acid treatment which is an improvement, as it forms an immediate coagulum, thus sealing off the tissues and aiding in preventing the loss of body fluids. It is no longer necessary to wait the usual forty-eight hours for a satisfactory coagulum to form.

Bettman[14] suggested the use of 10 per cent silver nitrate solution immediately after the application of 5 per cent tannic acid. This gives an immediate and satisfactory coagulum, and the author states that the silver nitrate is antiseptic. This method was used in a few of the following cases and is definitely an improvement over the use of tannic acid alone. It has the disadvantage of being somewhat painful for a short time after application and also provides a theoretical danger of argyria.

In 1936 a metallurgist burned his hand in an explosion of ferric chloride, and was given the first aid application of 5 per cent tannic acid. When he reached the hospital it was found that a perfect coagulum had

already formed and the burn was painless. Because of this he was allowed to leave the hospital at once and the hand healed without infection. This suggested a method

Table I
CAUSE OF BURNS

Metal	75
Natural gas	57
Gasoline	50
Chemical	49
Hot asphalt	36
Hot water	21
Crude oil	21
Steam	15
Electric	13
Miscellaneous	13
Total	350

which was applied to minor burns until its advantages were demonstrated. For two years it has been used exclusively on all burns. The method is as follows:

The burned area is first wet with fresh 5 per cent solution of tannic acid and immediately wiped with 5 per cent aqueous solution of ferric chloride. There forms an intensely black coagulum, which does not require dressing and gives the patient relief from pain. If vesicles later appear

Table II
CAUSES OF CHEMICAL BURNS

Cement	22
Sulfuric acid	12
Sodium hydroxide	6
Concentrated iodine	4
Creosote	3
Ammonia	2
Total	49

they are opened and the raw area treated the same way. If for any reason the first application does not give a satisfactory coagulum in all areas this may be obtained by reapplying the solutions. This decreases the difficulty of nursing and increases the patient's comfort by quickly relieving the pain and by removing the necessity for wet dressings and a wet bed. After the first dressing, the treatment is the same as that formerly used with tannic acid alone, depending on the seriousness of the burn and the symptoms.

On arrival of a burned patient at the hospital the dressings previously wet with tannic acid are removed, any vesicles present are opened, and the entire burn is

Table III
PERCENTAGE OF BODY SURFACE BURNED

No. of Cases	Percentage Burned
271	1 to 10
36	11 to 25
31	26 to 50
12	over 50

wet with 5 per cent aqueous solution of ferric chloride. The patient is removed to bed and covered with a cradle containing sufficient light bulbs to maintain a bed temperature of 100 degrees for the first forty-eight hours, after which the temperature is regulated for comfort.

Table IV
CASES IN WHICH TIME WAS LOST FROM WORK

No. of Cases (207)	Percentage of Body Surface Burned
128	1 to 10
36	11 to 25
31	26 to 50
12	over 50

The hemoglobin reading and erythrocyte count are obtained and intravenous infusion of 5 per cent solution of glucose in normal saline is begun. Arrangements are then made for transfusion. If there is a rise of over 10 per cent in the hemoglobin read-

Table V
CASES REQUIRING HOSPITAL TREATMENT

No. of Cases (107)	Percentage of Body Surface Burned
28	1 to 10
36	11 to 25
31	26 to 50
12	over 50

Total days in hospital—1,697
Average number of days in hospital 15.4

ing within forty-eight hours a transfusion is given. Morphine is repeated as necessary.

The nurse is instructed to reapply 5 per cent tannic acid followed by 5 per cent ferric chloride to any new vesicles or areas where a proper coagulum is not obtained on the first dressing. More than two applications are rarely required.

Within a few days symptoms of toxemia may appear. Vogt,[84] Robertson and Boyd,[72] and others have shown that a toxin is

liberated from burned tissue and that this is the cause of the toxemia. It has been stated[1] that the toxemia is due to massive infection with Streptococcus hemolyticus, and 1 per cent aqueous solution of gentian violet is used to combat the infection. The gentian violet is used in place of tannic acid, the further treatment being the same. This method has been used in a few cases

TABLE VI
DAYS LOST FROM WORK
(Exclusive of fatal cases)

No. of cases	207
Total days lost	5,728
Average no. of days lost per case	27.6

in this series but in no way has it shown itself superior to tannic acid and it has the disadvantage of staining the bed linen. Recently brilliant green[3] and other aniline dyes have been suggested for their bactericidal effect on both Gram-positive and Gram-negative organisms. My limited experience has shown that aniline dyes form a crust much more slowly than does the method referred to above.

TABLE VII
AMOUNT OF PERMANENT DISABILITY AT DISCHARGE
(Exclusive of fatal cases)

Disability	No. of cases
5 per cent hand	1
10 per cent hand	1
15 per cent hand	1
20 per cent foot	2
15 per cent arm	1
25 per cent arm	1
5 per cent total	1
15 per cent total	1
20 per cent total	1
Total	10

Infection is not uncommon in burns treated late with tannic acid or in cases where tannic acid has been applied after first dressings with greases or ointments; however, it is easily controlled by removing the crust over the infected area with scissors and applying wet dressings of Dakin's or normal saline solution.

In uninfected burns the crust is usually off within ten to fourteen days and it becomes necessary to repair the raw surfaces with skin grafts. This should be done at the earliest possible time as it hastens recovery and prevents the distressing contractures too often seen following severe burns. Pinch grafts will usually be found most satisfactory as they can be applied to larger areas and secured from smaller donor sites. These may become unsightly because of variations in pigmentation but this is not a contraindication when areas covered by clothing are concerned. For regions

TABLE VIII
MORTALITY

Per Cent of Body Burned	1 to 10	11 to 25	26 to 50	Over 50	Hospitalization	Totals
No. of cases	271	36	31	12	107	350
No. of deaths	0	0	4	9	13	13
Per cent mortality	0	0	12.9	75	11.9	3.7

about the face, either pedicle, whole thickness or split grafts are to be preferred, depending on the area to be covered and the donor area available.

The granulating bed is prepared by dressing with Dakin's solution, followed by a few days of dressing with compresses wet with normal saline solution, changing

TABLE IX
PERIOD OF DEATH IN FATAL CASES

No. of Cases	First Day	Second Day	Sixth Day
13	7	4	2

the dressings several times daily until the granulations have reached the proper condition. If pinch grafts are used, they can be applied earlier as an absolutely sterile field is not imperative.

The pinch grafts are cut very small and applied as closely as the donor site permits. After application they are covered with perforated cellosilk, two layers of sterile gauze and a layer of sterile sponge rubber half an inch thick. This sponge rubber is best obtained as bath mats and may be had at any department store. It should be washed with soap and water, thoroughly rinsed with water, dried and autoclaved. Following the application of the rubber

the whole part is firmly bandaged and not changed for eight days. The rubber absorbs the secretions and prevents the grafts from floating off. After eight days the entire dressing is removed and the area again dressed with compresses kept wet with normal saline. Occasionally, it becomes necessary to control exhuberant granulation between grafts but this is easily done by touching with silver nitrate or copper sulfate stick.

It has been shown[83] that there is a remarkable loss of blood serum from burns, reaching as high as 70 per cent of the total blood volume within twenty-four hours, and cases have been reported in which the hemoglobin reached 200 per cent concentration as a result of this serum loss. However, a concentration reaching 125 per cent is a critical level and immediate steps should be taken to correct it by transfusion or, if blood is unavailable, by infusion of saline and glucose. This concentration of blood increases the cardiac work required for circulation and, unless prevented or corrected in the early stages, will result in death. Therefore, intravenous infusion should be begun immediately after the first aid is completed. Hemoglobin readings should be taken every two hours during the first forty-eight hours following a burn, and if the blood shows more than 10 per cent concentration in spite of infusion of fluids, a transfusion should be given. If given early, a transfusion reduces the blood concentration as the plasma remains in the circulation; if given late, it is lost into the tissues and the concentration continues.

Electrical burns require special attention since their seriousness is out of proportion to their size. Such burns result in roast pork or charred areas, often quite small. Because of their size and relative painlessness they are often mistreated. They are usually deep, frequently extending to bone and they often destroy tendons and nerves in the area between the points of electrical contact. This results from the formation of a miniature electric arc which develops a temperature of several hundred degrees, depending on the voltage and the electrical conductivity of the contacts. It is time saving to cut away all burned tissue as quickly as a line of demarcation is formed and do such plastic repair as is indicated. These burns all require estimates of extensive temporary disability, and if not treated radically may result in permanent disability, depending on the structures involved.

Chemical burns may be treated as burns from fire except in their first aid. They should be washed copiously with water, the clothing removed and the burns washed again before applying first aid. No attempt should be made to neutralize the chemical as this results in a more severe burn due to the heat of neutralization penetrating still farther.

The following series of 350 cases consists of a group personally treated. It is reported to show the economic loss as well as the mortality of industrial burns. (Two hundred thirty-five of these cases were reported in a previous paper.[67])

It should be emphasized that the period lost from work is possibly shorter than average since most of these burns occurred in well conducted industrial plants where it is usually possible to secure light forms of work for injured employees. It is true that there is an intangible loss in working an employee at other than his usual occupation, but work lessens the employee's economic loss and improves his morale.

The estimates of permanent partial disability are made under the Industrial Compensation Laws of Texas and are probably not entirely comparable to estimates of disability made under the laws of other states since these laws are seldom comparable.

SUMMARY

1. The first aid care of industrial burns is discussed.

2. Certain fundamental differences of treatment of chemical and electrical burns are pointed out.

3. The economic loss resulting from burns is stressed.

4. Results obtained in 350 personally treated burns are reported.

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A CONSIDERATION OF SOME TRAUMATIC EYE CONDITIONS AND THEIR TREATMENT

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I SHALL endeavor in this consideration of traumatic eye lesions to take up the groups which are seen most frequently by those engaged in traumatic surgery and to outline briefly methods of diagnosis, prognosis and treatment.

I. FOREIGN BODIES IN THE EYE

A. Foreign Bodies in or on the Cornea. Since foreign bodies upon the surface of or imbedded in the cornea make up about 25 per cent of all eye injuries it seems expedient to discuss this condition in detail. The importance of a foreign body imbedded in the cornea depends upon the location and also upon whether infection with subsequent ulceration and scar formation results. Nearly all abrasions resulting from foreign bodies, if the foreign material be promptly removed, heal rapidly with no resulting scar. A foreign body imbedded in the center of the cornea which has been neglected or perhaps unskilfully removed may, on the other hand, lead to ulceration with scar formation which may cause marked impairment of vision and even 100 per cent permanent disability.

1. *Symptoms.* The usual complaint of a patient with a foreign body on the cornea is one of scratching, discomfort or even severe pain. The severity of the pain or discomfort depends largely upon the amount of ulceration developing from the subsequent infection.

2. *Diagnosis.* The diagnosis is usually easy and is made by direct inspection under rather high illumination. A very practical method to obtain this illumination is by the use of a pencil flashlight. In case the foreign body is very minute it may be necessary to use a magnifier such as a Berger loupe in order to locate the particle. Very occasionally the diagnosis of the presence of a foreign body is very difficult. This situation occurs when a minute body penetrates deeply and is covered by edematous corneal tissue which loses its transparency and obscures the foreign material.

3. *Prognosis.* The results from prompt removal of a foreign body from the cornea are usually good, for healing takes place rapidly and little if any scar remains. However, if such an injury is neglected and especially if the material is rather heavily infected, then ulceration may be rapid and extensive with scar formation varying from a slight opacity to a dense scar. Also if the affected area is directly in front of the pupil, marked diminution of vision often results with corresponding permanent disability.

4. *Treatment.* Prompt and thorough removal of invading foreign bodies under antiseptic precautions is the immediate procedure. A local anesthetic such as 2 per cent butyn, ½ per cent pontocain or 4 per cent cocaine instilled at five minute intervals three or four times gives sufficient anesthesia in most instances. A cotton wound applicator should be used in those cases in which the foreign material is superficial. A gentle to vigorous brushing of the area will often remove the material without injuring the corneal tissue. If the foreign body is imbedded and especially if it was hot when striking the eye, as often happens with emory wheel accidents, then it becomes necessary to use a sharp spud or curette to loosen and remove such bodies. If the corneal tissue is scorched it requires considerable time and patience to remove

all of this scorched tissue which acts essentially as foreign material.

In those cases where minute pieces of steel are imbedded very deeply near Descemet's membrane, it is often helpful to apply a weak magnet to the needle which is being used to free the foreign body, for by this method constant forward traction is exerted; otherwise the foreign body may be pushed backward into the anterior chamber. In a few cases the foreign body can be loosened but owing to the manipulation of the needle the corneal tissue becomes edematous and cloudy. The object may thus become invisible so that the removal has to be delayed to the following day at which time the cornea probably will have regained its transparency.

If the corneal epithelium or deeper tissues show abrasion, the point of injury should be touched with some antiseptic as 2 per cent mercurochrome, 1 per cent ethyl hydrocuprein or weak tincture of iodine in order to lessen infection.

5. *After Treatment.* The after treatment of corneal foreign body wounds is almost as important as the initial removal. If much trauma has occurred then a mydriatic should be ordered, providing the intraocular tension is normal. This procedure not only is the greatest factor in relieving pain but assists in the healing process. Hot compresses applied every three or four hours for ten-minute periods are also beneficial, for they increase the lymph flow to the cornea.

One almost absolute rule should be the examination of these patients after a twenty-four hour period, by which time the question of infection can usually be definitely determined. If pain has been rather severe and if the vessels surrounding the cornea are engorged, and especially if there is a whitish ring surrounding the point of injury (Fig. 1), then infection has definitely begun and prompt and energetic treatment should be inaugurated. Nearly constant heat should be applied, the pupil should be kept dilated and antiseptics frequently instilled. If the infiltration extends, then parenteral use of milk or other foreign proteins should be tried. Heat in the form of Shahan's thermophore is of great value.

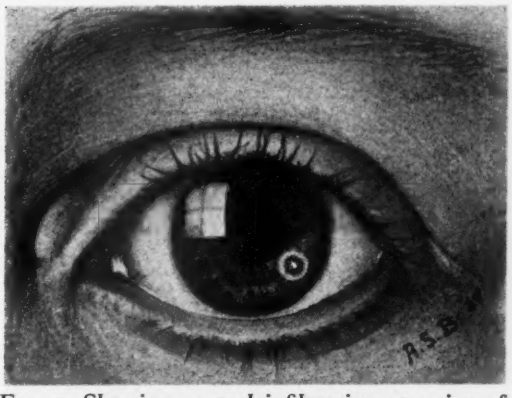

Fig. 1. Showing central infiltration at point of injury with a circular area of infiltration surrounding it.

Since these procedures are difficult to follow in the home hospitalization is usually necessary.

B. Foreign Bodies within the Eye. Of the various foreign bodies that penetrate the eye those of iron or steel are the most frequent. Fortunately these injuries are becoming much less frequent since greater care is exercised in the protection of the eyes in industrial work. A rather detailed history of the injury is essential in this group of patients, since it is important to know the direction in which the foreign body was traveling when it struck the eye, whether it was clean steel or infected material, and to have, if possible, an examination of the tools used at the time so that the probable size and composition can be judged.

1. *Diagnosis.* Usually there is little difficulty in determining the point of entrance and an *x*-ray with localization will show the position of the fragment, but occasionally after a history of injury there is little or no external evidence of trauma. If, however, there is a history of injury with unexplained symptoms a radiograph should be made, for occasionally a foreign body will be discovered. Occasionally too, the localization chart will be in error a few millimeters so that instead of the foreign

body being located within the eye it may be found to be just beyond the sclera in the orbital tissues.

2. *Treatment.* In case the localization shows the foreign body in the orbit rather than in the eye then conservative surgery indicates watchful waiting. Occasionally an orbital abscess results, but usually metallic foreign material within the orbit is tolerated without symptoms unless it has carried in a large amount of infection. In case a piece of steel or magnetic metal is within the eye, prompt removal should be undertaken. If the point of entrance is through the cornea and the lens has been damaged, removal through the point of entrance should be attempted, after which any points of prolapsed iris should be removed and a conjunctival flap thrown across the point of penetration.

In case the foreign body has entered the eye without injuring the lens, an exact localization should be done and the removal attempted at the point nearest the sclera providing this is in front of the equator. In case the body is behind the equator the magnet should be applied at some point in front of the equator nearest to the foreign body. The sclera should be incised, the magnet applied at the opening and extraction attempted at this point.

Since a detachment of the retina is an occasional postoperative complication, it has been suggested that an application of diathermy needles should be made surrounding the point of extraction in order to prevent this complication.

In a survey of the results of cases in which an intraocular foreign body had been removed, it was found that about one third of the patients retained good vision, one third useful vision and one third lost the eye. If a foreign body is left within the eye the eventual results are almost universally bad.

II. LACERATIONS OF THE EYE

With the advent of the motor car the number of cases of laceration of the cornea and other coats of the eye from flying glass have been greatly increased. Quite often these cases receive first aid in the emergency ward and by a general surgeon. Since the most important treatment is this same first aid, it is necessary to render adequate service at this time.

In case the cornea has been penetrated there is almost certain to be a prolapse of the iris. If this is found to be present a fine forceps should be used to catch up the prolapsed iris after which it should be cut away with fine scissors following proper cleansing of the eye. If the lips of the corneal wound are in good apposition with no tendency to gape, a small amount of bichloride of mercury salve (1:5,000) should be applied within the conjunctival sac and the eye lightly bandaged. If, however, there is a gaping of the wound one should not attempt to suture the edges of the wound, which is most difficult, but should cover the wound with a conjunctival flap, either after the method of Jameson or by means of a bridge flap. Either of these supports the edges of the cut.

It is usually unnecessary to remove the conjunctival flap, as the sutures loosen and the conjunctiva returns to its usual position. If this does not occur, owing to adhesions taking place between the conjunctiva and the lips of the corneal wound, it may be necessary to dissect accurately the conjunctiva from the cornea. This should not be done under ordinary circumstances for from ten days to two weeks.

If the penetration has gone beyond the cornea and anterior chamber then the lens becomes involved and we have a traumatic cataract with which to deal. In this case the treatment varies with the amount of penetration of the lens. If there is a large amount of soft lens substance present either in the wound or in the anterior chamber, immediate irrigation to wash out as much of the soft lens substance as possible should be attempted. The procedure must be accomplished with as little trauma as possible; otherwise a large loss of vitreous results. Following this procedure there is usually a considerable thickening of

the capsule and an opening through this membrane often becomes necessary. At times a traumatic cataract may even develop from a blow upon the eye which does not cause penetration (a contusion cataract). Even a blow on the head at a point removed from the orbit may cause a cataract to develop (a concussion cataract). Either of these conditions is handled by the usual methods for removal of soft cataracts.

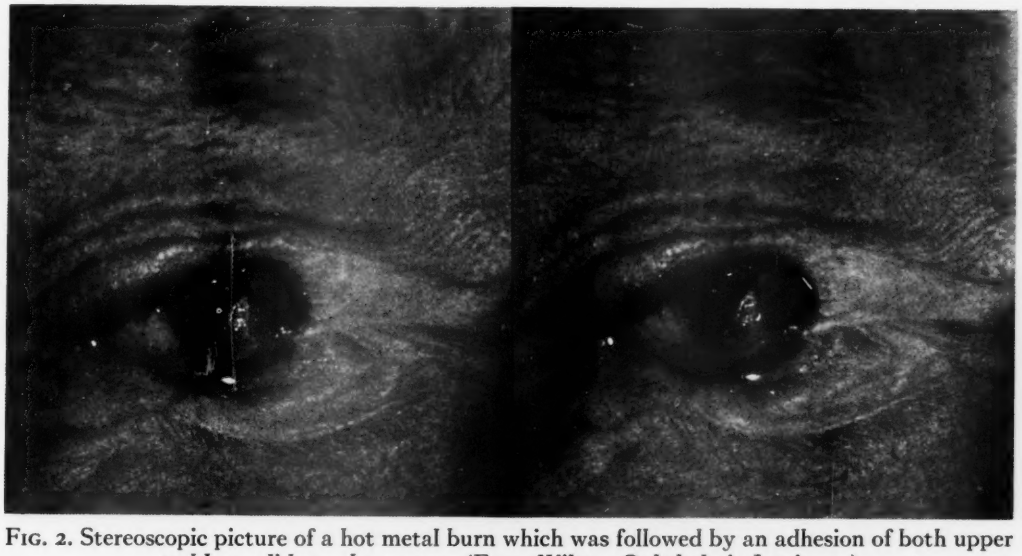

Fig. 2. Stereoscopic picture of a hot metal burn which was followed by an adhesion of both upper and lower lids to the cornea. (From Wilmer Ophthalmic Institute.)

III. BURNS OF THE EYE

A. Thermic Burns. While burns of the eye from heat are occasionally seen as a result of escaping steam or from hot metal, they are much less frequent than chemic burns. Minor burns of the cornea and conjunctiva occur rather frequently from such causes as a lighted cigar or cigarette and occasionally from a curling iron. The minor burns are quite painful but usually heal in a week or two under treatment with atropine and mild antiseptics. The burns from steam or molten metal may be very extensive and cases are seen where the cornea is perforated and the eye so damaged as to require immediate enucleation. If extensive burns of both the bulbar and palpebral conjunctiva have occurred the problem of restoring a normal functioning eye in most difficult. This type of a burn is frequently followed by a complete symblepharon or adhesion of the lids to the eyeball (Fig. 2), and at times the lids themselves become adherent to each other. The cornea also becomes opaque and vision is reduced to the point of motion or even light perception.

Treatment consists in the use of the frequent instillation of castor oil along with antiseptics. In some instances the use of cod liver oil seems to be more beneficial than that of castor oil. Whether or not the presence of vitamin A has any influence locally is open to question. The use of a contact glass placed over the cornea has been suggested to prevent ulceration of the cornea as well as to prevent complete symblepharon. A 1 per cent tannic acid jelly is also useful in combating the acute reaction. At times a plastic operation may be used to attempt the restoration of the lids and function of the eye. Often all efforts to keep the lids separated are without avail and various mechanical appliances placed in the conjunctival sac are forced out by the contracting conjunctiva.

B. Electric Burns. Burns of the eye from electric flashes are usually limited to irritative changes resulting from the excessive amount in the ultraviolet spectrum. The conjunctiva is injected and there is pronounced photophobia. The use of a colored glass to remove the excessive light

and the instillation locally of an anesthetic such as ½ per cent pontocaine until the symptoms subside is all that is usually required. If more severe burns occur the treatment should be symptomatic.

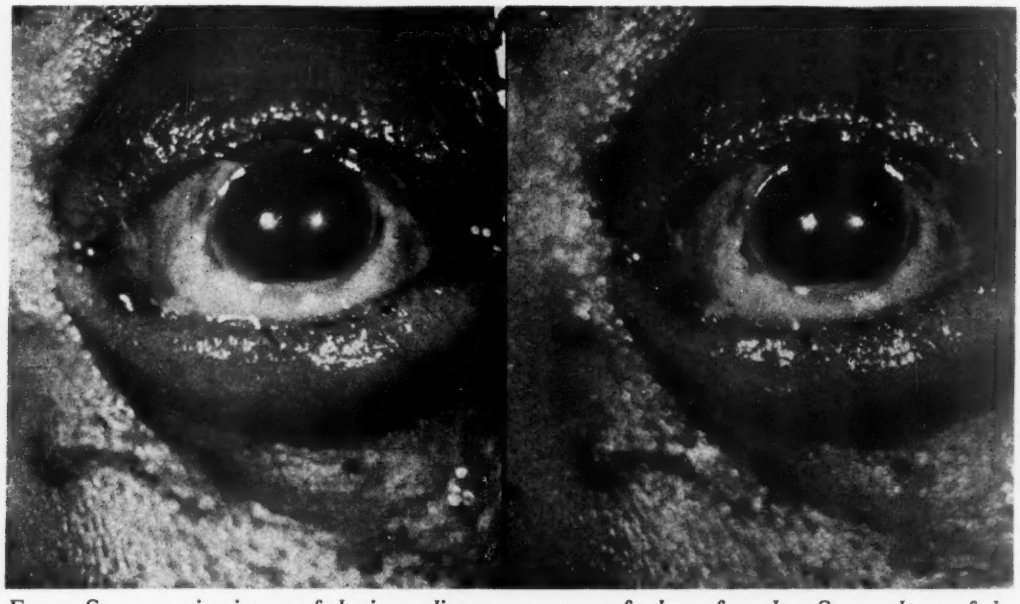

Fig. 3. Stereoscopic picture of the immediate appearance of a burn from lye. Some edema of the cornea is present, the conjunctiva and sclera are bleached. Both eyes were lost from ulceration with perforation.

C. Chemic Burns.

1. *Acid Burns.* Acid burns vary from slight to severe. The most frequent accident causing this condition is that of a spark exploding the hydrogen mixture which is given off in a charged battery. The explosion usually forces some of the fluid into the eye with a resulting burn. The reaction is usually quite severe with some opacity of the cornea resulting.

Treatment should consist of flushing the conjunctival sac with large quantities of water, but neutralization with an alkali should not be attempted since the reaction is as a rule much worse. If the cornea is affected the pupil should be dilated with atropine and circulation stimulated by hot compresses and the instillation of 1 per cent dionin three times a day.

2. *Alkali Burns.* Alkali burns are usually more severe than the acid ones as in the former the coagulum seems to be limited while in the latter the effect extends more deeply. In the immediate treatment the eye should be irrigated with large quantities of water and in this condition neutralization with a 2 per cent acetic acid or vinegar seems to be helpful. Later the use of 1 per cent tannic acid jelly as well as a mild antiseptic should be employed. The difficulty in keeping down adhesions applies here as well as in the case of thermic burns. In case the alkali is lime then the use of 10 per cent neutral ammonium tartrate solution is of value.

In all of these chemical burns, however, too sanguine a prognosis should not be given since often the eyes are much worse after the end of a week than they are immediately after the accident (Fig. 3), or even after a day or two.

CONCLUSION

Finally let us consider those cases which may be safely handled by the traumatic surgeon as distinguished from those that should have the attention of an ophthalmologist.

Foreign bodies in the conjunctival sac and those upon or even imbedded in the cornea should be treated by the industrial surgeon. A foreign body that has abraded

the cornea should be referred to the ophthalmic surgeon since only a slight extension of an ulcer with subsequent scar formation may cause marked loss of vision with a correspondingly high percentage of permanent disability.

All cases of intraocular foreign bodies should also be referred to the ophthalmic surgeon, as I know of no condition requiring greater judgment than is required in the handling of this group.

In cases of laceration of the cornea from flying glass, first aid should be given by the general surgeon, but if the lesion is extensive it would seem to be wise to pass the case to the ophthalmologist who must take the responsibility for the loss of vision which almost universally results.

Burns of the eye should be given very prompt treatment. This can and should be performed at the first aid depot, but again if the burn is extensive it requires hospitalization under adequate ophthalmic supervision since a great amount of permanent disability as well as deformity is apt to result.

Adhesive plaster skin traction is, as a rule, neither reliable nor adequate for the treatment of fractures of the shaft of the femur in adults.

VISUAL ACUITY: ITS RELATION TO THE FORM SENSE AND THE APPLICATION OF THIS RELATIONSHIP TO MEDICOLEGAL PROBLEMS

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IN 1925 the writer read a paper[1] in which the fundamental principles of the Snellen acuity notations and the mathematical relation of these notations to usefulness of macular vision were discussed. In the present paper the writer from another point of view undertakes to elucidate further some of these underlying principles and to point out their practical application to the medicolegal problem of evaluating partial losses of vision.

That there is need from a medicolegal point of view for a further consideration of the problem of evaluating partial impairment of vision is particularly apparent from the confusion resulting from the lack of unity in the method of computation among the industrial compensation boards of many states—a condition due largely to the false opinions of some ophthalmologists appearing as experts and to the erroneous views of some writers who hold that acuity notations are fractional parts of vision.

The state of confusion and chaos which exists among the Industrial Compensation Boards of different states is shown by the following facts:[2] first, that in the twenty-three states which have adopted some method or schedule by which partial loss of vision may be determined there are eighteen different methods of computation; second, there are not more than four states which are in substantial agreement in method; and third, for identical degrees of visual acuity the evaluations by the different state industrial commissions often differ greatly. For example, when visual acuity is reduced to $^{20}/_{25}$ Snellen, New York State makes an award of 20 per cent, whereas several states make an award of 5 per cent, and some make no award, considering $^{20}/_{25}$ as no loss. For $^{20}/_{40}$ New York awards 50 per cent, some other states 10 per cent, a difference of 40 per cent. Furthermore, this lack of unity in evaluating vision and the errors incident thereto lead directly to injustice both to those who carry the economic responsibility for payment of damages and to those who have suffered some permanent partial ocular disability. In the latter class are both those whose eyes have been damaged as a result of industrial employment, and those who may have subnormal acuity of vision from other causes. In the first instance, those states making the higher awards are placed under an economic disadvantage in competition with neighboring states. In the second instance, employees looking for jobs are being excluded from employment, and employee advancement is handicapped when management is led to believe that minor degrees of subnormal acuity greatly reduce ocular efficiency, as, for example, to regard one as 50 per cent blind or incapacitated who has $^{20}/_{40}$ acuity of vision. The acceptance of a single method for evaluating defects in vision is most desirable in the interest of unity and of justice.

The lack of a uniform method of evaluating partial visual disabilities is due mainly to two causes: (a) the erroneous interpretation of the Snellen formula, and (b) the failure to solve the problem of the proportionate loss of function in its relation to each acuity notation. The error in interpretation originates in the assumption that since the Snellen acuity notations have the appearance of fractions they express equivalent fractional parts of the essential

function of the form sense: and also in the idea that the term "visual acuity" can be regarded as synonymous with the "act of vision." The failure to solve the problem of *proportionate loss* of *function* for each acuity notation was due to the fact that the mathematical relation between acuity notations and loss of function had not been established prior to the conclusions which are embodied in the report of the Committee on Compensation for Eye Injuries of the American Medical Association.[3] A correct evaluation depends on an understanding of the meaning of acuity notations and also of their relation to the impairment of the function of the form sense which is associated with such notations.

In order to make clear the nature of the function of the form sense, the following fundamental principles of the physiology of vision should be kept in mind. Vision primarily is a light and form sense; the latter, being dependent on sufficient light stimulus to make form distinguishable, is inseparable from it. In this paper adequate light is assumed. The form sense concerns the ability of the eye to perceive objects; and from this ability of the eye to recognize adjacent points of retinal sensations as separate, there is evolved the principle of the "minimum separable." Thus, from this ability to discriminate separate objects, or their parts as separate, there is evolved not only the function of the perception of form but also the function of perception of *detail* of form. This latter function is known as the resolving power of the eye. Resolving power, then, is the faculty or function of the form sense by which discrete objects are perceived as separate and detail of form is recognized.

The problem in medicolegal cases involving partial loss of vision is *the determination of the percentage loss of functional values*. To determine this percentage loss there must be an understanding of the relation between visual acuity and resolving power. These terms, visual acuity and resolving power, are often, though incorrectly, used synonymously. Both concern the form sense; both are measured by visual angles: however, there is a fundamentally different concept inherent in the terms which it is important to understand in solving the problem of evaluating visual disabilities. The term "visual acuity" is often used in a metonymic sense since it may convey not only the idea of sharpness, which is its literal meaning, but also it may convey the concept of function. From common usage and experience one may associate with the meaning of the term "acuity of vision" the entirely different concept of ability or power of eye to accomplish an act, a function which is associated with known acuities. However, the literal meaning of the term "visual acuity" does not include the concept of function, since acuteness or sharpness of vision is a quality or characteristic which does not signify action; whereas *resolving power* does indicate *action*, a necessary characteristic of physiologic *function*. By keeping in mind this concept of functional ability, that is, visual resolving power in its proper relation to visual acuity, all partial visual disabilities can be more easily comprehended and the problem of their evaluation simplified. The solution of the medicolegal problem of determining the percentage loss of functional values will be found in *understanding* the relation of acuity notations to resolving power.

Before discussing this relationship the nature and significance of the Snellen notations should be considered. These have been discussed in the paper mentioned above and are briefly reviewed here. The Snellen notations, although recorded in the form of fractions, are *symbols* expressing relative visual acuities. Professor Snellen in no instance regarded the term "visual acuity" as being synonymous with the "act of vision." The symbol *V* (originally Snellen used the symbol *S* for "Sehschärfe") signifies *acuity* of *vision*. Snellen[4] did not reduce these notations to common fractions, nor did he regard them

as representing equivalent proportional parts of vision. He disapproved of this proceeding, stating that it was "unfortunately followed by many," and that this reduction "tends to produce a wrong impression of the proportional values of the visual acuity which are indicated by the test." The notation of visual acuity d/D is an equation or ratio which expresses the measure of the visual angle by *comparing d* with *D*. The notation above the line indicates the distance at which the test is made and the notation below the line indicates the distance at which the test letters subtend an angle of 5 minutes. It should be observed that in every visual acuity notation, according to Snellen's own definition of this equation, the size of the normal visual angle is *compared* with the size of the smallest distinguishable visual angle of eye being tested, the first measurement being placed over the second measurement. Therefore, simply from the nature of the Snellen notations they are not fractions but are ratios. There is no more justification to regard the acuity of $^{20}/_{40}$ as half blindness than it is to regard a person one-half dead who has his blood pressure notated as 80/160, or that five cents compared with ten cents is one-half dollar because $^{5}/_{10} = {}^{1}/_{2}$. The following further consideration of acuity notations will demonstrate that they do not partake of the nature of fractions. Fractions are equal or aliquot parts of unity, therefore, the unit or whole must be divided into equal parts to produce fractions. The expression $^{20}/_{20}$ acuity is universally accepted as a complete unit or the whole of visual perception; now, as visual acuity (or perception) decreases this unit quantity, the 5 minute angle, is not divided but is constantly compared with a larger angle. Thus, all acuity notations are not fractions but are ratios by which one quantity is compared with another.

To show that a judicial body after due consideration of voluminous testimony comprehended that acuity notations do not express fractional parts of vision a few apposite decisions of the Supreme Court of New York are appended. In the decisions quoted below the question at issue before the court in four suits related only to the question of the interpretation of acuity notations. In each case the court held the same opinion as is expressed in the following abstracted quotations.[5]

"It will be seen, however, that the denominators, in the various symbols, when used as fractions, denote units, such as objects and angles, which are never the same. The denominators, then, cannot subdivide the constant unit of perfect sight or the constant unit of blindness. Consequently, the symbols cannot be fractions of sight or blindness."

"The assumption has no support in testimony or in reason."

"It is apparent that the assumption leads to absurdities which contradict and disprove the theory.

"The Industrial Board in making the award assumed that the Snellen symbol $^{20}/_{100}$ signified the retention of 20 per-centum of vision. . . . The assumption that $^{20}/_{100}$ Snellen has the significance of a common fraction, or that the Workmen's Compensation Law by virtue of the section quoted, Sec. 15. Subd. 3, has stamped $^{20}/_{100}$ Snellen as industrial blindness, has no support in reason or in authority. (Struble vs. Vacuum Oil Co., 210 App. Div., 344; S. C. 214 ed. 844.)"

"The weight of authority doubtless inclines to the rules adopted by the American Medical Association."[6]

Then, if acuity notations are not fractions of vision, the issue upheld by this court decision, could they by any other possible mathematical consideration be found to express an equivalent proportional loss of vision a contention advanced by some? To point out that acuity notations which are comparative ratios do *not* likewise express proportional parts of resolving power the following two considerations should suffice. First, when the assumption is made that acuity notations are equivalent fractional expressions

of vision, we shall find that an eye having an acuity of $^{20}/_{10}$ (such acuity is not uncommon) being compared with the standard normal would have a $^{2}/_{1}$ ratio; that is, such an eye would have 200 per cent central vision and should such an eye by industrial accident or otherwise be reduced to $^{20}/_{20}$, it would sustain a loss of 100 per cent when it was reduced to normal—obviously an absurdity.

Secondly, again on the above assumption, there will be found another absurdity by making comparisons of the percentage differences in functional ability between the successive gradations of acuity ratios in the geometrical progression series of Green. This series of test characters constructed with the constant increase for each gradation of 1.26 is the accepted minimum and practical degree of gradation which can be resolved for each successive increase in the visual angle throughout the entire range of qualitative vision. It is indisputable that the better the vision the better is the eye able to resolve the same or smaller differences between successive gradations in acuity. This must be true also when these same gradations are expressed in terms of functional value. (Functional values can be expressed best in percentages.) The better the vision the better able is the eye to resolve increasingly smaller *percentage* differences between successive gradations.

The converse is also indisputable that the poorer the vision the less able is the eye to resolve the same or larger differences between successive gradations; and this also must be true when these same gradations are expressed in terms of percentage of function. However, if we compare the percentage differences of acuity notations between successive geometrical gradations, when the acuity notations are reduced to equivalent fractions, we shall find that, for each gradation which the eye is able to resolve, there is a constantly smaller percentage difference between gradations as acuity falls. For example, an acuity between $^{20}/_{20}$ and $^{20}/_{25}$ indicates a 20 per cent difference in resolving power, whereas an acuity between $^{20}/_{33}$ and $^{20}/_{40}$ indicates a 10 per cent difference, showing that resolving power is twice as good between $^{20}/_{33}$ and $^{20}/_{40}$ as it is between $^{20}/_{20}$ and $^{20}/_{25}$. And for each successive gradation the percentage difference is constantly less. Thus, when visual acuity is good the eye has relatively poor resolving power and when acuity becomes poorer it has relatively better resolving power. This may be stated in another way; when central vision is good it is comparatively less efficient than when it is poorer. Thus, a mathematical consideration of this series with the assumption that acuity notations are fractional parts of vision is proved to be a reductio ad absurdum hypothesis. To illustrate these observations Table 1 is presented in which there are also set forth comparative values between this method and that of the A. M. A.

Attention is called to a third, an experimental consideration, which proves that acuity notations are not equivalent fractional parts of vision. Such experiments have been made and the results published.[7] A series of obscurants were constructed which had the property of reducing acuity by a definite amount. Among these several were selected which reduced visual acuity to $^{20}/_{40}$ and these were superimposed before the eye in series of one, two, three, four, five and six and their effect on acuity determined. It is obvious that if $^{20}/_{40}$ acuity is 50 per cent vision or a 50 per cent loss of vision, then if one such obscurant reduces vision from $^{20}/_{20}$ to $^{20}/_{40}$ acuity, two exactly similar obscurants superimposed together before this eye would take away the remaining 50 per cent and thus completely destroy all vision or all resolving power. If one such obscurant renders the eye one-half blind, two exactly similar ones superimposed before the eye would necessarily make the eye totally blind. However, this was not found to be true. Two $^{20}/_{40}$ obscurants reduced acuity to $^{20}/_{65}$ and it required six of these superimposed together to reduce the form

sense to zero resolving power. Thus, resolving power for the entire range of detail vision is divided into six equal steps or divisions by a uniform scale, and therefore, a $^{20}/_{40}$ reduction in acuity does not reduce the whole of central vision one-half, but only one sixth. Each $^{20}/_{40}$ obscurant did reduce resolving power approximately 16.6 per cent and not 50 per cent. Thus, this experimental method proves that acuity notations do not express equivalent proportional parts of resolving power.

There are many recognized authors, among them Weiss,[8] Green,[9] Landolt,[10] Donders,[11] and Fuchs,[12] who have pointed out the erroneous interpretations which follow the reduction of Snellen acuity notations to equivalent fractions, approaching the problem from a mathematical and physiologic point of view. Contributions by these authors were made before workmen's compensation was established, thus refuting the statement that until the advent of compensation laws the Snellen tests were regarded as setting forth the percentage loss of vision. Other authors, among whom are Magnus,[13] Wurdemann,[14] Hansell,[15] Holt,[16] Allport,[17] Chapman,[18] Sharp,[19] and Smith,[20] later studied the problem of evaluating vision, taking into consideration also an economic point of view. All found that the acuity notations did not express an equivalent fractional earning ability or of usefulness of vision.

These early authors pointed out the errors which followed the reduction of acuity notations to fractional equivalents but did not definitely set forth their *true functional values*. The later authors made an advance in solving this problem of evaluating vision and did compute values for acuity notations, but these are not entirely in agreement in all details since different methods were used. The differences in values for like acuities computed by these authors are not very great but are important. The desirability for removing these differences by the establishment of a uniform method for evaluating partial visual disabilities which should have a scientific foundation (one in which empiricism should be removed as far as possible) was recognized by the ophthalmologists of the American Medical Association. A committee was appointed in 1919 for this purpose and after several years of study, investigation, experimentation and research a final report was submitted and approved by the A. M. A. in 1925.[21]

The most difficult part of the problem presented to this committee in evaluating vision was the determination of the relation of acuity notations to resolving power. Its solution was found by research and experimentation which demonstrated the effect on resolving power caused by the addition of one or more similar obscurants. These obscurants were used as a unit scale for measuring the influence on resolving power by observing their effect on acuity. The effect of the use of this scale was: first, it produced a definite and constant increase in the visual angle and thus caused a constant and a proportional decrease in resolving power; and second, it demonstrated that the relationship between resolving power and the corresponding increase in visual angle is in substantial accord with Weber's and with Fechner's psychophysical laws and also in accord with the mathematical expression of Lambert's law of light absorption. The deductions which were made from this study were that "equal increases in the visual angle correspond to equal proportionate reductions in visual efficiency," or, in other words, "the visual efficiency of an eye diminishes in a constant ratio for successive equal increases in the visual angle," or "equal increases in the visual angle reduce the visual efficiency by a constant ratio." This constant was found to be 0.83625 for each increase of one minute in the visual angle (or for each increase of 5 minutes in size of test characters): that is, each successive increase of visual angle by this amount reduces the resolving powers of an eye 83.6 per

cent of what it was before such increase. By the use of this constant a table of visual efficiency has been made showing the relation of acuity notations (size of visual angles) to resolving power. This table is contained in the report of A. M. A.*

It should be observed that by this method every equal increase in the visual angle—every injury to the eye causing an equal reduction in acuity—has exactly the same proportional effect on resolving power, no matter what the acuity was before such injury; so that for equal degrees of reduction in function (for equal amounts of ocular disability) exactly the same percentage loss of function is determined. For example, if an eye sustains an injury in which acuity is reduced from normal to $^{20}/_{40}$ (an increase in visual angle of 5 minutes) the resolving power is 83.6 per cent of what it was before such an injury—83.6 per cent of 100 = 83.6 per cent, the percentage efficiency for $^{20}/_{40}$; if visual acuity was $^{20}/_{40}$ before an injury and was then reduced to $^{20}/_{60}$, the percentage loss of efficiency would be again 83.6 per cent of the $^{20}/_{40}$ efficiency factor, or $.836 \times .836 = .699$ or 69.9 per cent; for $^{20}/_{80}$ efficiency it would be $.836 \times .699 = 58.5$ per cent; etc. The resolving power is constantly reduced by 83.6 per cent each time the visual angle or test letters are increased 5 minutes. Thus, for the same increase in visual angles, for identical degrees of damage to function, identical percentage losses are established. This is not true of any other method for evaluating visual loss. It seems obvious that this method by which the same percentage evaluation can be made for the same amount of injury is absolutely fair, just, and reasonable. It is based on sound scientific principles.

Field vision can be given only brief consideration here, but since it also is related to the form sense, attention is called to a few important facts. The form sense is derived from all parts of the retina and every part of the retina has a similar function, differing only in quality; resolving power is a function of the entire retina, although it functions most efficiently in the macular area. The perception of motion and position are predominantly the function of field vision (peripheral vision). Therefore, resolving power is determined by measuring visual acuity centrally, whilst the extramacular form sense is determined by measuring the area of its presence, or absence. It is not necessary to determine either resolving power or visual acuity outside of the macular area since either is best measured where it is predominant. Neither is it necessary to measure the form sense centrally since the test for visual acuity determines its presence or absence. The relative values of central acuity and peripheral acuity may be determined by consulting the familiar computation of Dor,[22] but this would only complicate the problem and would not alter the final evaluation of either resolving power or the peripheral form sense.

The method for determining the loss of visual efficiency as adopted by the House of Delegates of the American Medical Association embodies the conclusions presented by the investigations mentioned above for both the central and the peripheral form sense. For a period of six years the committee on Compensation for Eye Injuries studied the problem of evaluating visual disabilities. The final report embodies not only the conclusions of the different members of this committee but also of other interested persons, economists, mathematicians, sociologists, and ophthalmologists, who were consulted. And now after a period of thirteen years, this report has received an almost universal approval as being founded on sound scientific principles. However, these principles are not yet fully understood by some ophthalmologists and by others having legislative or administrative authority. Based on misunderstanding of these principles the following are some of the

* For all details of the experiments, observations, conclusions, and mathematical formulae and deductions see previously published papers.

criticisms of, or objections to, the A. M. A. report in its application to medicolegal cases, especially in compensation cases; (1) the use of the terms efficiency, usefulness and resolving power; (2) the weighing of the near acuity test; (3) economic considerations which it is claimed modified the efficiency computations; and (4) the applicability to various state laws of the principles promulgated in the A. M. A. report. A brief discussion of these items follows.

1. *Terminology.* Efficiency as commonly defined means "competency in performance, ability to produce results" (Century dictionary). In physics it is more precisely defined as "the ratio of work accomplished to energy expended." Efficiency was defined as "the ratio of the resolving power of the eye under consideration to the resolving power of the normal eye," the measure of energy being represented by the size of visual angle. Resolving power is expressed in terms of efficiency, and usefulness is the percentage value of this function which equals the work accomplished. Usefulness and efficiency are used synonymously. The evaluation of resolving power on the basis of "*competency in performance*" of its function would seem to require no argument to justify it. The evaluation of the form sense, or of resolving power on the principle of efficiency or usefulness is sound scientifically and has the weight of authority.

2. *Weighing near Acuity.* The weighing of the near acuity test was proposed and advocated by some commissioners of industrial boards. It was contended that since in industry most work was done at the near point, near vision was much more important than distant vision; therefore, the test of visual acuity should be done at the working distance. The validity of this argument was conceded and the relative importance of good near vision was given a twofold value compared with distant vision. The purpose in recommending the weighted near test was to establish thereby a more just and more liberal item of computation for ocular disability than would be computed when the distant acuity only was considered. It was not the intent of this recommendation to cause a reduction in efficiency as has been contended by some. In the practical application of this method the effect has been usually to increase the percentage loss.[23] The near test, despite its difficulties, adds to the accuracy of the evaluation of visual efficiency and should generally be done. At times the near test is unsatisfactory and misleading, due usually to lack of coöperation on the part of the examined, which makes it unreliable. Every ophthalmologist should use his individual judgment in use of this test unless it is designated by statute. It might be desirable to use the near test when employment is at the near point and the distance test when employment requires distant vision.

3. *Economic Considerations.* The efficiency tables in the report express the exact mathematical measure of the function of the macular form sense which was determined by the application of those physical and psychophysical laws relating to light and to the physiology of vision found to be apposite. The computations were in no degree altered or modified by the consideration of any economic principles. However, in the introductory paragraph preceding the acuity table there is a statement that an acuity of $^{20}/_{200}$ was found to represent 80 per cent loss of macular vision. Also in a later report of another committee of the A. M. A., economic blindness is defined as an acuity of $^{20}/_{200}$. Therefore, the statement made in an address before the I.O.U.A.B.C.[24] in Washington, D. C. to the effect that according to the report of the A. M. A. $^{20}/_{800}$ represents industrial blindness is entirely erroneous.

4. *Applicability of A. M. A. Report.* Although the report on compensation of eye injuries is based on the principles of total disability (blindness being regarded as a total disability), this does not

TABLE I

PERCENTAGE RELATION BETWEEN VISUAL ACUITY NOTATIONS AND LOSS OF FUNCTION FOR EACH SUCCESSIVE STEP IN THE GEOMETRICAL PROGRESSION SERIES OF GREEN, COMPARING VALUES AS COMPUTED BY REGARDING ACUITY NOTATIONS AS FRACTIONS AND BY THE A. M. A. METHOD

A	*B*	*C*	*D*	*E*
20 over 20	100.	100.	0.0	0.0
20/20 to 25	80.	95.6	20.	4.4
20/25 to 34	62.5	90.3	17.5	5.3
20/33 to 40	50.	84.1	12.5	6.2
20/40 to 50	40.	76.5	10.	7.6
20/50 to 63	31.6	68.	8.4	8.5
20/63 to 80	25.	58.8	5.6	9.2
20/80 to 100	20.	49.4	5.	9.4
20/100 to 125	16.	39.8	4.	9.6
20/125 to 162	12.1	30.	3.9	9.8
20/162 to 200	10.	20.	2.1	10.
20/200 to 250	8.	8.	2.	12.

A. Visual acuity notations in the geometrical progression series of Green, each successive increase in test characters being 1.26 times the preceding.

B. The indicated percentage of central vision when the latter of each of these notations in *A* is reduced to equivalent decimals.

C. The percentage resolving power for each successive progression as determined by the A. M. A. method for computing functional values.

D. The difference in functional ability for successive gradations for column *B*.

E. The difference in functional ability for successive gradations for column *C*.

render the separate parts or sections inapplicable to the laws of those states where compensation is based on total or partial losses of *one* eye. The report contains a method for computing the functional loss of each eye separately and when the valuation of loss of use of only one eye is desired that may be computed without considering the effect of such loss on the coördinate action of both eyes. However, when medicolegal questions arise relating to the effect on vision when both eyes are involved the report sets forth correct principles by which such disabilities may be evaluated.

Some progress towards uniformity in evaluating visual loss has been made during the past decade. Ten states and also the United States government have adopted schedules based on the fundamentals contained in the report of the committee on Compensation for Eye Injuries of the A. M. A. And eleven other states have adopted schedules which do not greatly differ from the group mentioned above in their percentage schedules of visual loss, although they differ in some essential details. For example, some require that visual acuity tests shall be made without the use of glasses, an unjust and indefensible rule. New York State is the only state where awards for visual disability are based on the principle of regarding visual acuity notations as equivalent fractional parts of vision.

There still remain twenty-three states which have not adopted any schedule or method whereby visual losses may be computed. In these states the evaluation of visual disabilities is left to the examiner's opinion or to the discretion of the industrial commissioners based on the report of the examination of the ophthalmologist.

The relation between visual acuity and resolving power of the form sense has been established on a mathematical basis. An understanding of this relationship will remove present lay confusion and medical controversy in medicolegal cases involving the evaluation of visual disabilities.

SUMMARY

Vision is a light and form sense. From the ability of the eye to discriminate detail of form is evolved the function of resolving power.

The problem in medicolegal cases is the determination of the percentage loss of functional values. To determine these percentage functional disabilities, the relation between visual acuities and the associated functional losses must be established on a mathematical basis.

The term "visual acuity" does not include the concept of function. Acuity notations are not fractions of vision nor are they fractional equivalents of resolving power.

Comparing the successive gradations of acuity notations in the geometrical pro-

gression of Green and assuming that acuity notations express fractional ability of the form sense, it is shown that the better the acuity the poorer is functional ability—when vision is good it has relatively poor resolving power. This disproves the assumption.

A method for computing the functional and proportionate value for acuity notations has been established. By this method each equal increase in the visual angle reduces visual resolving power (efficiency) by the same percentage.

By application of this method to the medicolegal problem of evaluating visual disabilities, for identical degrees of damage to function identical percentage losses are established.

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THE DIAGNOSIS AND TREATMENT OF THE RUPTURED URETHRA

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WHEN considering rupture of the urethra and the location of the lesion it is necessary to remember our land marks. In order to do this we need a mental picture of the anatomy, blood and nerve supply, and divisions of the urethra. With this in mind we shall briefly try to refresh our memories.

As we know, the urethra extends from the bladder to the external meatus. It is S-shaped, about 8 inches in length, and is composed of three parts: the prostatic, the membranous, and the spongy portion. Clinically we have an anterior and a posterior urethra, the anterior divided into the bulbous and the penile, while the posterior corresponds to the membranous and prostatic urethra. Entering the verumontanum is an elevation of the urethral mucosa produced by the ejaculatory ducts and utricle. The prostatic urethra is about 1¼ inches in length; the membranous extends from the prostate to the penile bulb and is ½ inch in length. Cowper's glands are located just behind the junction of the membranous with the spongy urethra. The spongy or pendulous urethra is about 6 inches in length, is embedded in erectile tissue, and terminates at the external meatus.

The prostatic portion of the urethra derives its blood supply from the middle hemorrhoidal and the inferior vesical arteries. The membranous portion is supplied by the middle hemorrhoidal and the transverse perineal artery, while the bulbar artery supplies the spongy urethra. The dorsal vein of the penis returns the blood from the anterior urethra into the plexus of Santorini. The superficial perineal and dorsal innervate the penis.

There are five points of narrowing in the urethra: at the internal sphincter, in the membranous urethra, at the penoscrotal junction, at the penonavicular junction, and at the external meatus.

The urethra is lined by columnar epithelium which extends to the meatus and fossa navicularis where it becomes squamous. There are three layers, mucous, submucous, and muscular.

Rupture of the urethra includes all contusions and lacerations of this organ and may be brought about by injuries to the canal inflicted from without or within. As a result one may have to deal with urinary obstruction, extravasation of urine, or fistula.

There are two types of rupture: external and internal. External injuries may be brought about by puncture wounds, contusions, and lacerations of the urethra, some of the common causes of which are gunshot wounds, stab wounds, blows, falls, or crushing injuries with or without fracture of the pelvis. O'Connor reported eight cases of rupture of the bulbous urethra from falling astride a man-hole cover. Internal urethral injuries may be brought about by a tear following passage of an instrument, insertion of a foreign body, or, in some rare cases, by passage of a rough calculus. False passages may be induced by improper instrumentation.

Rupture of the urethra is of four types: rupture of the pendulous pouch; rupture of the bulbous portion anterior to the triangular ligament; rupture of the bulbomembranous portion anterior to the triangular ligament; and rupture of the membranoprostatic portion at, or posterior to, the triangular ligament. Rupture of the pendu-

lous portion is very rare. Rupture of the bulbous or bulbomembranous portion is caused by accidents, as falling astride wagon tongues or fences or by a kick in the perineum; this is the most common type. Rupture of the posterior urethra is usually due to fracture of the pelvis, and seldom occurs without it. Incomplete rupture of the urethra may leave the mucosa intact; a partial rupture tears the mucosa from the urethral wall; in a complete rupture, the urethra is divided.

Patients when seen with this condition present these important symptoms: pain, tenderness, bleeding, partial or complete retention, and tumor formation. The pain at the time of rupture is very severe. Tenderness exists primarily at the point of injury; later on there may be swelling, induration, and extravasation. Bleeding from the meatus is a common symptom. The disturbance of urination may vary from hesitancy caused by pain in the milder cases to complete retention in the severe cases, brought about by laceration or injury to the urethra, and in some cases to spasm of the cut-off muscle. If not speedily relieved, the retention becomes intensified and extravasation may develop. Tumor formation is primarily due to hemorrhage; later on it may be due to infiltration and suppuration of the soft tissues.

The diagnosis of rupture of the urethra is not nearly so difficult as is the exact localization of the lesion. If there is complete rupture it is impossible to pass a catheter, and this may also be true even in milder cases. In severe cases of rupture of the urethra accompanied by shock the *x*-ray will determine the presence of fracture of the pelvis. If the patient is unable to void and a catheter cannot be properly inserted, the diagnosis is more easily determined.

In transverse wounds there is free hemorrhage, and if the urethra is severed there is retraction of the segments with resulting extravasation of urine. If this is not properly handled, fibrous tissue may form and stricture develop, and there may be partial or complete retention. The spongy tissue and the mucosa are torn and contused, giving rise to hemorrhage; not only are the corpus spongiosum and the urethra injured, but the fibrous coat of the spongiosum and the last layer of superficial fascia are also affected.

The development of these symptoms varies according to the extent of the injury. In the milder cases there may be no more than temporary pain, slight swelling, and the appearance of blood in the urine. In the more severe cases urination is very painful, there is marked swelling with ecchymosis at the site of the injury. If the patient is not catheterized or attended surgically, extravasation, infection, and sloughing may develop, accompanied by chills and an elevation of temperature. When this condition exists, if surgical interference is neglected, the skin over the region becomes swollen and dark red in color, crepitation follows, and gas forms in the tissues. If the condition is allowed to continue the infection becomes so extensive that the scrotum may slough away, leaving the testes bare and exposed with drainage of urine and pus from the openings. In the majority of cases this proves fatal.

TREATMENT

The patient should first be relieved of pain. A specialist should be called if in a rural district, no matter how simple the condition may appear. If there is bleeding from the urethra there should be consultation, not only for the patient's sake but for the protection of the doctor as well. We must realize that a failure in diagnosis often means a failure in treatment, and one failure will cause the public to forget hundreds of good results. If the patient can void and there is only a small amount of hemorrhage he should be watched carefully and expectant treatment used. If the patient cannot void an attempt should be made to pass a catheter; if the attempt is successful the catheter should be fastened in. In profuse hemorrhage transfusion and surgical interference are

immediately indicated. Following operation glucose should be administered intravenously if necessary.

In complete rupture of the bulbous urethra the types of operation, according to Higgens, are: end-to-end anastomosis; insertion of catheter (Rutherford technique); suture of roof of the urethra. End-to-end anastomosis and suture of the roof of the urethra mean perineal section, and this must be very carefully done because of the risk of infection. If the wound becomes infected and breaks down we may have a more dense stricture than if we introduced a catheter and packed the wound with gauze (Rutherford). In this latter type of rupture immediate surgical intervention is necessary.

Intrapelvic Rupture. The author has had three cases of this type, each patient being badly shocked with rupture due to pelvic fracture. In each of these cases the urethra was not only ruptured but macerated. The bladder could easily be outlined.

The same procedure was used in each case: a suprapubic cystostomy was done and profuse hemorrhage was encountered in the space of Retzius. A catheter was passed into the urethra until diverted by the rupture, another catheter was passed from the bladder to the rupture, the ends sutured together and one catheter drawn down through the penis. As the catheter seemed too short a large rubber tube was sutured to the remaining catheter and then gently pulled through the penis, after being well-lubricated. The rubber tube was 3 feet long, about the size of a 26 F. catheter. Openings were made in that portion of the tube lying in the bladder and each end of the tube placed in a receptacle. This was done so that the bladder could be irrigated two ways. The bladder was closed around the tube with No. 2 plain catgut, one suture was placed through the bladder and tube, and a black silk suture was placed through the skin and tube to hold the tube in place. The tube not only acted as a splint, but gave the surrounding macerated tissue an opportunity to heal without coming in contact with the bladder secretion. The tube was left in for about fourteen days. In each of these cases the patient seemed to come through with simple dilatation and the urethra repaired itself.

Stricture, extravasation of urine, infection, and finally, gangrene may follow in untreated cases if not handled properly.

Differential Diagnosis. It is important to differentiate between rupture of the posterior urethra and rupture of the bladder. This can be done if the bladder can be outlined.

Postoperative Treatment. The patient should be relieved of pain, and should receive symptomatic treatment. Fluids are forced; a nourishing diet is given; proper elimination is assured. In order to prevent stricture the patient should be followed for at least a year and occasionally dilatation should be done.

During the World War I saw ten patients with rupture of the urethra: one of the pendulous urethra, six of the bulbous, four of the intrapelvic type.

The rupture of the pendulous urethra was due to shrapnel which caused fracture of the right thigh and numerous lacerations in addition to the injury to the penis. There was no laceration of the penis but marked contusion and ecchymosis. The patient was seen within four hours from the time of injury; he was bleeding slightly from the meatus and could not void; fortunately I had a rubber catheter at my aid station, but only with difficulty was I able to get the catheter in the bladder and remove about 500 c.c. of urine. The catheter was left in and the patient sent down the line. In three cases of rupture of the bulbous region I was able eventually to introduce a catheter. The other three were rushed back to a field hospital as rapidly as possible.

The four cases of rupture of the intrapelvic type were seen while I was attached to a base hospital. These patients were badly wounded and had fractured pelves. One died on arrival at the hospital. In

each of the four cases the bladder could be outlined. It is unfortunate that these patients could not be followed.

Since the war four cases of rupture of the urethra have come under my care. One of these is briefly given here:

The patient, a little boy of 4, complained of severe pain in the pelvic region after being hit by a truck. Dr. B. of Annapolis called me at my office in Baltimore the morning following the accident. His description was so typical of rupture of the urethra that I asked him to get the operating room ready and assured him I would be there within the hour.

The child weighed 43 pounds, was well-nourished, with flushed cheeks, but he had an anxious expression and complained of severe pain in the lower abdomen. The abdomen was markedly distended, dulness was discernible up to the umbilicus, and bladder dulness could be made out. Marked contusions were present over the lower right quadrant and upper right thigh. The lower extremities were normal. An *x*-ray revealed fracture of the superior ramus of the pubic bone, position fair.

A midline incision was made extending from just below the umbilicus almost to the symphysis; bleeding was controlled as encountered. The bladder was exposed and found to be intact. There seemed to be marked bulging just below the space of Retzius. As the dissection was gently carried down, there was a gush of blood which had been under pressure and had distended the soft tissues about the pelvis. The bladder was opened and a catheter was then passed down the penis to the site of rupture; it was impossible, however, to get the catheter into the bladder. Another catheter was passed through the vesical orifice to the rupture; the ends of the two catheters were sutured and the lower catheter was entirely withdrawn; perforations were made in the portion of the catheter lying in the bladder, in order to permit irrigation of the bladder.

The bladder was closed with plain catgut, the fascia with chromic, the skin with black silk; two silk sutures were placed through the skin and the tube. Since the patient was a small child the catheter was long enough to be used instead of a rubber tube. The ends of the catheter were placed in a receptacle, a drain was inserted in the space of Retzius, and the catheter was left in place for fourteen days.

The patient had an elevation of temperature for about five days, but only on two occasions did it go above 103. He voided on the eighteenth day and has been voiding since that time with no apparent difficulty or diminution in stream. The child is normal and healthy and appears never to have had anything wrong with him.

CONCLUSION

1. The anatomy of the urethra is summarized.
2. Types of rupture are indicated.
3. The importance of calling a specialist is mentioned.
4. Importance of hospitalization, operation and surgical interference is emphasized.
5. Ten cases were seen during the war.
6. Four cases of intrapelvic rupture were treated by the author, using suprapubic operation with through-and-through tube as splint and openings in the tube in order that the bladder could be irrigated.

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EFFECT OF TRAUMA AS "PRECIPITATING" FACTOR IN CERTAIN NEUROLOGIC DISEASES

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SO many people have a sneaking belief in telepathy! In that each man is a microcosm of the culture of the race, so there must lie in each man a deep stratum of belief in magic.

Under affliction, and in distress, the distraught personality reaches down to this vestigial remnant of the occult, and says feebly "after all, there must be something in it." How many of us are there who have thought of some almost forgotten friend, and within a week of this unusual occurrence, received a message from that friend? One remembers on seeing the letter his recollection of the friend a day or so before, and perhaps reaches the quite unjustifiable conclusion that he thought of the friend because the friend, at that moment, was thinking of him. Such a man lacks controls in his experiment, for he does not remember the great accumulation of occasions when he has thought of a friend and received no message at all.

One sometimes thinks that of all the professions, Medicine, which clings to the skirts of the sciences, is the least scientific. No physicist, no astronomer, and certainly no chemist, would ever think of reaching general conclusions from so paltry a number of special instances as we so constantly do. We spend, as regards the occasions of which we have been speaking, perhaps forty years accumulating evidences of a positive character and neglecting all evidences opposed; therefore we come to conclusions, or wished-for speculations which are, if not absurd, at least unwarranted. Almost no body of men so easily falls a victim to *post hoc, propter hoc* reasoning as ourselves, and especially perhaps in the matter of the relationship of injury to disease.

One notices that the influence of injury as an etiologic factor in the "precipitation" or production of organic diseases of the nervous system, is chiefly stressed with regard to those nervous ailments the etiologic cause of which is entirely unknown.

When a textbook for students is written, and the possible causes—and almost nothing in nature is impossible—of "schizophrenia," manic depressive psychosis, Parkinson's disease, multiple sclerosis, and degeneration of the spinal anterior horns and the pyramidal tracts in the spinal cord, are under review, no author has the courage, his ignorance of facts being completely thorough, to leave out injury as a possible fountain and origin of evil.

The Great War, which exposed many millions of men to all kinds of stress and strain and structural injury, was a laboratory in which many of these hypotheses could be tested. For example, consider the problem of injury as being capable of precipitating or causing multiple sclerosis. This disease is very common in the people of Northern Europe. It is one of the most usual nervous diseases in the wards of British hospitals. It has a very clear age incidence. Should it appear before seventeen or eighteen years of age, it is fair enough to question the accuracy of the diagnosis; and should it begin after forty-five, the same doubt is legitimate. The British armies in France, in a four year period, must have included in that time between three and four million men; these men belonging to the race susceptible to multiple sclerosis fell almost entirely within the age incidence of that disease. They were exposed to a high degree of injury and stress. But in several years' service in

France I saw but two cases. Were injury a really important cause of multiple sclerosis there surely should have been at least a small epidemic of that disease.

The same may be said of the great psychoses. I mean by these, dementia praecox or "schizophrenia," and manic depressive psychosis. These two abnormal states represent the bulk of the patients in our state hospitals. No man living knows their cause. Almost every textbook, cautiously thinking in the subjunctive mood, says or suggests that they may be produced, or more vaguely still, precipitated, by injury. In France, among fighting troops, they were rare. A total incidence of insanity in cases of "gunshot wounds of the head," according to British official records, is the same as that for the general male population, which varies from 0.34 to 0.35 per cent. I understand that experts attached to the German and French equivalents of the British Ministry of Pensions, have stated that they had no evidence other than the cases of actual dementia and mental deterioration after damage to the brain, to suggest that psychoses followed "gunshot wound—head."

Statistical experience shows that psychoses in general, and "schizophrenia" in particular, were no more frequent among fighting troops than in the same number of civilians in peacetime. We do not know the cause of "schizophrenia" but the great experiment of the war certainly gives us no authority for the idea that this condition can result from injury.

The substitution of Greek for Latin in naming this pattern of abnormal behavior —which has no appearance of being a disease in its own right—is an index of our intellectual progress during the last decade in knowledge concerning it.

It is surely absurd to be told that Tom Jones, having fallen off a ladder, consequently developed "schizophrenia" six months later, unless one is also told that a thousand or ten thousand Tom Joneses, sustaining much graver structural and emotional injury, did *not* develop "schizophrenia."

The literature of this subject teems with little names who report one case, or five cases, in support of the idea that nervous diseases, of unknown etiology, have been brought on as a result of some specified accident. It is better, on the whole, to regard the opinion of persons whose work one knows, and whose honesty and sense of proportion one respects. So it is noteworthy that Kinnier Wilson, in 1923, could not accept injury as an etiologic factor in any of the progressive nervous deteriorations, nor in cerebral tumor.

Otto Veraguth gave a comprehensive report on this matter in the International Neurological Congress at Berne in 1931. He says, "The common trauma-causing World War brought with it no increase in general paresis or tabes (he quotes a number of names, Gaupp, Glieniberger, Moser and others); nor were multiple sclerosis, nor the diseases of the Parkinson complex (Kehrer, Souques, Roussy, Lhermitte), nor were the muscular atrophies, nor syringomyelia noticeably increased." As has been said, Wilson has stated that no increase in brain tumors could be found as the result of tens of thousands of brain injuries.

Terbrüggen,[1] writing from the Pathologic Institute of the University of Greifswald, says that it was perfectly demonstrated that, during the war general paresis did not increase, but decreased. He goes on to say that no war experience would support the idea of an aggravation of an existing paralysis. "In general," he continues, "paresis, during the years of the war, had a course no more rapid than previously."

Jahnel[2] considered that statistics of war injuries, and also of injuries in civil life, gave no indication of any increase in paresis as a result of injury, and, in reviewing individual cases, he believes the fact is too often overlooked that acceleration of general paresis often occurs without injury at all.

Robert Bing,[3] discussing head injuries as a factor in the pathogenesis in paralysis agitans, expressed his opinion that if

patients and their histories were carefully enough examined, the number of individuals in whom Parkinson's disease could at all be laid at the door of injury, would be reduced to a minimum.

On the other hand, Naville and de Morsier, discussing the medicolegal aspect of Parkinson's disease with a history of accident, drew the conclusion that this condition might be caused by contusional encephalitis. They noted the appearance of a striatal syndrome several days to three months or more after such head injury as a cerebral concussion. Their account, however, lacks figures.

And Veraguth[4] concludes that whether direct injury to the basal ganglia or injuries which cause circulatory disturbances in that region can result in post-traumatic Parkinsonism, has not been definitely demonstrated. However, as occurred in a case of my own, if a man sustains a serious fall and concussion, with fractures of the long bones, and, *within three or four days*, develops a dystonic torticollis of organic type, it is fair to believe that there must have been a causal relationship between the accident and the immediately subsequent diseases.

In all of these matters, it is not enough to quote possibilities; only probabilities should be considered. And as has been said, the probabilities are scarce. Perhaps one might speak here of our use of the word "trauma." Too often we use a Greek word instead of an English word to allow ourselves, if not a toying with intellectual dishonesty, at least a less rigid perception of actual truth. So with "trauma." Were we always to say "injury" where we habitually say "trauma," we would immediately invite a challenge to define the injury. One would immediately say, "What injury?" "Trauma" is a conversational blank check to be filled out by the hearer, and may mean much or almost nothing. The more we know of virus infections, circulatory disturbances, vitamin lacks, errors in oxygen supply, the less, I believe, we shall hear of injury, either as "precipitating" or causing degenerative nervous ailments or the great psychoses.

As regards the role of injury and epilepsy, W. W. Wagstaffe[5] refers to Percy Sargent who stated that "up to the end of the year 1920, the Ministry of Pensions had traced 18,000 cases of gunshot wound in the head, of whom 4½ per cent were subject to epileptic fits."

Wagstaffe analyzes the total number of 377 cases all of which came under his own care in France during the years 1916–1918, and all of which were followed up. The statistics show that the vast majority of cases of traumatic epilepsy are those in which there have been penetrating wounds of the head; in these the incidence of traumatic epilepsy is as high as 18 per cent. In all the rest the incidence is only 2 per cent and the incidence in the whole series is just under 10 per cent. He concludes that the incidence of traumatic epilepsy is definitely associated with the severity of the primary wound. Common sense, as cool as this, is rare.

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FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM AS SEQUELAE TO TRAUMA

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FOLLOWING an accident certain individuals remain ill for an unduly long period with physical or mental symptoms, or both, local or general in nature, or both, out of all proportion to the severity and extent of the injuries sustained. The disability is not due to post-traumatic structural changes in the parts injured and in many instances all physical evidence of injury has long since vanished. Yet pain in previously injured parts is bitterly complained of for many months, in spite of the fact that the physician cannot find a suggestion of local pathologic changes with the diagnostic means at his disposal. This is the most frequently encountered post-traumatic situation with which physicians have to contend, and it is all the more baffling and disconcerting to the physician because sooner or later he must express a definite opinion in compensation and liability cases as to the reason for the prolonged complaint of pain, its exact physiopathogenesis, the prognosis, and the percentage of disability. This monosymptomatic clinical picture of intractable pain in a previously injured part is as applicable to the foot as to the head, or, for that matter, to any part between.

The enigma of the chronic pain situation applies to headache in cases of head injury. It is my experience that in the majority of such cases the symptom is a functional one rather than the result of structural changes within the cranial cavity. In making this statement I have in mind the very large group of refractory cephalalgias continuing for months after a blow on the head unassociated with evidence of brain injury or where concussion symptoms were suggestive but equivocal. Functional cephalalgia is almost universally localized to the site of the blow, and radiates to cover half of the head on that side. It is associated with a point of extreme tenderness within the area injured, usually the scar if the scalp was lacerated. The unremitting character of the pain picture is strongly suggestive of its functional nature. Wide radiation of pain is often described, with extension into the posterior cervical region on both sides, or only to the side where the injury was received, across the shoulders into one or both limbs, or extending over the entire half of the body ipsilateral to the side of the head injury. Aside from these special characteristics of the functional type of post-traumatic headache there is difficulty in differentiating organic from psychogenic cephalalgia without closely surveying the entire symptom complex. Pain in the eyeballs, across the forehead, in both temples, at the nape of the neck and a constricting band encircling the head at the base are described alike in the neurotic and the organic states. Their differentiation is based largely upon a critical survey of the surrounding bill, so to speak, and not infrequently one finds himself confronted with a dual mechanism combining the organic and psychogenic. Although the neurotic head distress is characteristically unfavorably influenced by many extraneous factors, we must not lose sight of the fact that organic cephalalgia also is characteristically aggravated by exposure to direct sunlight, change in position of the body, prolonged physical and mental effort, etc.

Chronic backache following back strain is another troublesome pain situation running a respectable second to headache in the gamut of traumatic disabilities. Sooner or later the chronic back case becomes a medicine ball in the hands of the plaintiff

and defendant physicians, chiefly because too much stress is laid on negative *x*-ray findings. A very large majority of cases of back strain or sprain shows no radiographically demonstrable structural alteration. Lumbosacral joint twist and sacroiliac strain are all too common results of apparently trivial physical incidents, and in spite of negative *x*-ray findings the victim may suffer a very painful and protracted disability. Ligamentous, fascial and periosteal tears occur with sudden movement of the stooped body while lifting which precipitates an acute back disability with no confirmatory radiographic evidence. The ensuing symptom complex is not difficult to evaluate in the ordinary case; further discussion of this condition is not the main purpose of this paper. I am interested in the patient who has a protracted and irregular course as a result of nervous factors other than the changes wrought by the back injury.

A patient with such a condition complains of unremitting pain across the lower back, in some instances with radiation up the center of the back to the occipital region. He must walk cautiously, with the aid of a cane, lest "something slip out of place and send an electric shock to my head and floor me." When the original injury is unilateral sacroiliac, the ipsilateral lower limb is guarded lest "the knee buckle on me and I go down." Crutches are reluctantly discarded after many weeks' use in favor of two canes, later one cane. The same is true of the body brace, the usefulness of which had long since ceased to exist, and in many cases of doubtful necessity from the start. Such a patient is unwilling to bend his back in any direction. The manner in which he bends forward reflects his hypervigilant attitude, viz., the body bends at the hips, the spinal column retains its erect curves and the head is held rigidly in its original erect position. In most instances the spinae erector muscle masses in the lumbar region have normal and even tone, but in the more disturbed patient these muscles may be markedly contracted by his voluntarily maintained erect and stiff back.

Marked tenderness over a more or less wide area of the lumbosacral portion of the back is a common functional manifestation, or the point of "exquisite" tenderness may be limited to the originally traumatized area. Quantitative discrimination of pressure is not characteristic of this tenderness since the patient often squirms the moment he becomes aware of the examiner's finger touching the skin. After the demonstration of a rigid vertebral column in its entirety the patient may be observed to bend his back in a more or less normal manner as he reaches for his stocking on the floor. Similarly, after redirection of the patient's attention by questioning or by other tests, the examiner's fingers may dig deeply into the allegedly painful area of the back without producing a dissenting word or movement. This description of a common symptom complex does not exclude the existence of a focal basic organic defect, but it points to the functional overlay or hysterical epiphenomenon.

The post-concussion status may be organic or functional and the clinical picture may be all the more confused because of the similarity of the manifestations. The symptom complex of the concussion aftermath is well-known, but for better differentiation between the organic and functional I may enumerate the following characteristic subjective phenomena: headache, dizziness, impaired vision (asthenopia), head or ear noises, dulness of hearing, poor mental concentration, insomnia, mental and physical fatigability, emotional instability, forgetfulness, irritability, intolerance of noise, strong light and heat. Most patients have a number of these symptoms in varied combinations, but rarely do we encounter all of the symptoms in any given case. The neurotic individual may have any one or a number of these post-traumatic symptoms, and their proper identification as functional or organic is at times almost impossible.

There are individuals who, after suffering a head injury, anticipate the persistence of headache, dizziness and nervousness because of the common knowledge of these symptoms among the laity, and this attitude of auto-suggestion serves that individual's purpose advantageously when compensation or litigation is involved. The neurotic individual may say he feels dizzy, but he rarely describes the postural type of vertigo related to structural intracranial changes. He uses superlatives in describing his headache, leaving no doubt that his suffering is most intense and unremitting. He will trace the distribution of the pain from the area of the blow to various other parts of the head one at a time until he has covered the entire territory, and then describes the general devastating effect of the headache attack. When pressed for a more critical description of his dizziness, the patient frequently associates this symptom with his headache and does not differentiate between the two.

Asthenopia is variously described as inability to read because of blurred print or wavy lines on the page, dimness of vision, spots before the eyes, inability to discern objects at a distance and even double vision when tests of muscle balance reveal no defects. Color blindness is a rare condition following head injury; in one of my patients it was part of a severe hysterical state including stuttering, headache, insomnia, vomiting and falling. Because he could not differentiate the red from the green traffic light signals, he was snatched several times from the path of automobiles. Psychotherapy for five weeks removed the entire symptom complex, including the color blindness, after it had been present for three months. Unilateral tinnitus aurium and impaired hearing are rarely of neurotic origin and in these patients there is usually supportive evidence of an organic type of dysfunction.

Insomnia is something we cannot question unless the patient is under observation in a hospital. We often hear the allegation that the patient has not slept a wink since the accident, perhaps six or more months previous, but his appearance belies the statement. Nocturnal restlessness, wakefulness, slowness in getting to sleep or early awakening are nearer the truth than no sleep at all, but there is much room for exaggeration and dramatization in this regard should the patient be so inclined. The symptom of insomnia has no diagnostic value in either direction, functional or organic, and is accepted at its face value only to be interpreted in the light of the surrounding clinical picture.

Impairment of memory and hearing are frequently erroneously referred to by relatives when it amounts to no more than inattentiveness because of introspective preoccupation. Alterations of mood and dispositional changes are frequently observed, usually in the form of depression and irritability. In the mild reaction the patient may seem depressed because of his concern over his condition, but in the more severely disturbed individual a marked depressive state may result from the psychopathologic influence of anxiety. Hypersensitivity to external stimuli, both physical and psychical, results in behavior generally described as irritability. An individual so affected rebukes or abuses his children for the noise they make in his presence—noise which previously had not bothered him.

Neurasthenic or psychasthenic manifestations are quite common and cover the full range of functional nervous symptoms occurring in all types of psychoneuroses. General weakness, a feeling of exhaustion, rapid fatigue on exertion, and great effort necessary to get out of bed in the morning are phrases characteristically used by these patients to describe their general state of ill-being. Simple mental tasks, such as reading, making minor calculations, or engaging in discussions requiring decisive conclusions may bring about a state of confusion, mental fatigue and headache. The patient usually misconstrues this condition and speaks of it as faulty memory, when, as a matter of fact, it is evidence of fatigabil-

ity at the intellectual level and impatience because of emotional irritability. The emotional coloring of the psychasthenic picture varies with the type of individual, ranging from the garrulous paranoid to the tearful martyr; the former with venomous invectives places the blame for each and every symptom and pain at the feet of either his employer, the other participant in the accident or society-at-large; the latter tearfully describes his distress in the setting of the accident dramatically painted and relived in its every detail.

Another group of functional disorders following trauma is identified as major hysteria or conversion hysteria wherein there is displayed gross or objective disturbance of function at various levels, such as apparent loss of consciousness, motor paralysis, sensory defects and gross functional loss in the field of the special senses and the bodily viscera. Occasionally the patient or his relatives describe attacks of fainting and falling to the floor, with or without convulsive motor signs, which suggests the possibility of a post-traumatic epilepsy. The probable organic basis for such a condition following a head injury should always be given precedence in the diagnostic considerations, but after head injuries there are numerous examples of hysterical epileptoid attacks which are amenable to psychotherapy. Such an interpretation should not usually be given to an attack which savors of the more or less typical epileptic convulsion, although I have seen frothing at the mouth, facial twitching and rigid extremities in the psychogenic attack. Loss of sphincter control should be considered a diagnostic deterrent in favor of the organic until the course of the condition indicates otherwise. The more irregular and bizarre the episodic display the more likely are the factors psychogenic. Focal twitching of one side of the face or the distal portion of an extremity, intermittent or more or less constant, carries with it a focal cortical inference even in the presence of a hyperemotional outburst.

Two kinds of falling attacks without loss of consciousness and without convulsive movements are encountered in the post-traumatic psychoneurotic individual; one is concerned with an injured lower limb which suggests to the patient periodic giving way of the knee or hip joint, and the other type is related to postural dizziness following head injuries. The patient whose leg gives way anticipates such recurrences in association with intensification of the pain and he may believe that something slipped out of place in one or another joint, resulting in loss of balance and falling. These patients not infrequently suffer secondary injuries as a result of the fall, showing that they occur precipitantly and without deliberation on the part of the patient.

In the case of the vertiginous fall, the individual usually pitches headlong to the ground after he had been stooping for some minutes or longer while engaged in some minor activity such as weeding the garden or the like. Superficial injuries about the face and hands are also sustained in these falls, indicating the suddenness of the loss of static control. It may be argued that organic changes in the vestibular pathways probably explain this type of static control loss because the patient has post-concussion symptoms of vestibular character, yet in my experience this symptom occurs in the emotionally distressed patients and under circumstances which clearly indicate the psychogenesis of the disturbance. It should be kept in mind that a victim of Ménière's disease may fall suddenly in an attack; similarly, organic traumatic changes may explain the fall in some cases. However, I am simply referring to the fact that the symptom of postural dizziness occasionally suggests falling to the psychoneurotic patient.

Total or segmental paralysis of a previously injured extremity is quite a common hysterical display and it may or may not be associated with more or less complete anesthesia of the affected parts. An injured finger may remain rigidly immobile or the

entire hand may be held in a paralytic posture. An injury to the shoulder may be converted into a complete paralysis of the entire extremity or it may be limited to the shoulder and arm with preservation of power below the elbow. Paralysis of the upper limb is more common than that of the lower extremity insofar as complete loss of power is concerned; however, an injury to a lower limb frequently suggests a gait disturbance from simple limping to grotesque distortion on a pair of crutches. The sensory loss is generally segmental, ending in a sharp line either at the wrist, upper forearm, mid-arm or encircling the shoulder. Usually all forms of deep and superficial sensations are affected, but in many cases only touch, pain and temperature recognition is lost while vibration and position are preserved. In milder forms of hysterical anesthesia only pinpoint perception is gone and recognition is limited to blunt contact. Not infrequently a seeming paradox in these cases occurs in the form of anesthesia dolorosa, i.e., complaint of severe pain throughout the paralyzed extremity and at the same time complete loss of pinpoint sensation covering the entire integument of the affected part. A variety of disagreeable dysesthesias may accompany the hysterical sensory-motor paralysis, such as numbness, tingling, prickling, burning, coldness, electrical shocks, cramping spasm, and formication.

Unilateral blindness may be alleged after a superficial eye wound has healed and ophthalmoscopic examination discloses normal media and eyegrounds. Bilateral amblyopia or amaurosis occasionally follows conjunctivitis from irritative substances, and in the partially blind patient the characteristic finding is tubular visual fields. The same condition exists in some cases of head injury. With the removal of the disability by suggestive therapy the hysterical character of the involved mechanism is established. Attention should be called to the fact that occipital injuries may traumatize the visual centers with resultant impairment of vision and the ophthalmoscopic examination disclose no evidence of structural changes. The optic discs will show pathologic changes only when the intracranial pressure is elevated or when the visual pathways are directly injured anterior to the primary optic centers in the interbrain. It is an accepted fact that diplopia disappears with the closure of one eye, and when a patient insists that he still sees double with the previously injured eye when the good eye is closed the examiner must either question the patient's credibility or consider the symptom an hysterical one. Specific patterns of visual field defects, be they bitemporal or homonymous hemianopsias or homonymous quadrantanopsias, exclude hysteria as a basis for the visual loss because of their physio-anatomic significance.

Traumatic fright experiences sometimes bring about a speech disturbance either immediately or shortly after the accident. This may manifest itself in aphonia or one of several forms of dysarthria, such as stuttering, stammering, phonetic or tonal changes. A woman seated next to the driver of a car observed the setting of the impending collision, and a moment thereafter, although not the least bit hurt, stepped out of the car and stuttered when she was asked her name and address; the stuttering continued for three months. Shortly after the accident she developed tachycardia just as she had two years previously following a similar accident at which time she had had no difficulty with her speech. A chauffeur suffered a mild cerebral concussion when he fell from a soap box and struck the back of his head on the cellar floor, but had no speech difficulty until he was informed that he would not be reëmployed. His speech became monotoned, staccato and explosive with increasing crescendo toward the end of each sentence. This is the same patient referred to above in reference to color blindness. All of his symptoms were almost simultaneously removed after a number of weeks of intensive psychotherapy.

Symptoms of autonomic dystonia are not lacking by any means in the scheme of post-traumatic hysterical reactions, to wit, dryness of the mouth, lacrimation, globus, dysphagia, tachypnea, tachycardia, cardiospasm, emesis, pyrosis, belching, eructation, abdominal cramps, constipation, diarrhea, frequency of urination, coldness of the extremities with hyperidrosis. Marked weight loss often follows in the wake of both organic cerebral damage and psychoneurotic reactions following trauma. In general, the greater the loss of flesh in the functional state the more serious is the psychopathologic disturbance. When there is no weight loss it generally bespeaks a better prognosis.

Foster Kennedy is of the opinion that when headache and dizziness persist for more than four months in a man under 60 years of age, they are to be regarded as neurotic symptoms if the following seven criteria of head injury sufficient to produce organic changes in the brain are all absent —Roentgen evidence of skull fracture, bloody spinal fluid, bleeding from skull orifices (especially from the ears), focal cerebral palsies, convulsive states proved to be post-traumatic, ventricular distortion proved to be post-traumatic, and a history of prolonged unconsciousness. This succinctly stated opinion is attractive and heartening to one frequently confronted with such problems because a negative neurologic examination cannot be utilized as an all-exclusive diagnostic basis for differentiating a neurotic disability from an organic one. One cannot overstress the importance of a thorough-going clinical investigation in these cases before an opinion is expressed as to the physiopathogenesis of the symptom complex, its treatment and prognosis. In the face of entirely negative physical and mental survey the clinician must learn to evaluate each symptom in the light of the whole setting. The type of individual, the accident situation, the head injury data and special studies as mentioned in Kennedy's criteria, the compensation and litigation values and not least the pretraumatic socio-economic status of the individual, and his employment record must all be given consideration.

Granting the practical validity of Kennedy's opinion expressed above, it should also be pointed out that even with one or more of the criteria of brain injury in the history of any given case, the last post-traumatic sequelae may be entirely neurotic, the organic symptoms having subsided. It is by no means an easy matter to recognize the dividing line or the time when the organic manifestations cease and the neurotic ones begin, because they are intimately interlaced. As a rule the greater the emotional coloring of a symptom the greater is the probability of its being neurotic in origin. On the other hand, certain types of individuals may be so emotionally disturbed over correlated factors and circumstances that basic organic phenomena may appear to be more or less hysterical in nature. Therefore, one should not be too hasty in his identification and classification of the component parts of the subjective symptom complex and reserve this feature of his diagnostic considerations until the completion of the thorough clinical investigation. The combination of organic cerebral changes following trauma and psychogenic phenomena of a reactive kind is, in my opinion, more frequently the case than the existence of what might be called a pure psychoneurosis in patients who had sustained unequivocal injury to the brain.

The physician's prime interest in his diagnostic approach to this problem should be a careful neurologic examination with every aid available to him. It goes without saying that he should make every effort to settle in his own mind the question of whether or not the patient suffered a brain injury, its character and severity, by close scrutiny of the data in the anamnesis and hospital records which are available. Only on premises built on such data can one feel confident in his evaluation of the clinical findings. In the period of sequelae

we are concerned with the nature and extent of residual organic defects. Accordingly, appropriate clinical studies should be made. A radiolucent line indicating a linear fracture merely confirms the history of a head injury, but adds nothing of importance to the survey of residual pathologic changes in the brain; conversely, the absence of radiographic evidence of skull fracture does not preclude the possibility of existing cerebral organic changes. Perimetric studies may disclose significant sector defects which may be discoverable only with the color test objects while the form fields remain full. Enlargement of the blind spots with diminished visual acuity may exist with full form fields. Tubular vision is generally conceded to be an hysterical phenomenon, whereas, concentric contraction of both fields may be either functional or organic, but in the latter case it is to be interpreted in the light of other organic indications. In the case of diplopia, muscle balance tests will serve to confirm or disprove the organic basis for the double vision and identify the affected cranial nerves.

Where vertigo is a prominent symptom, tests for vestibular function (Barany) are an invaluable aid to establish the fact that there is a pathologic change in the vestibular pathways. Labyrinthine lesions may be quite reliably differentiated from intramedullary or cerebellar defects. Spinal manometry occasionally brings to light the existence of chronic intracranial hypertension or, less frequently, hypotension, following brain injuries. In this type of case the symptom complex may have all the features of a psychoneurotic disturbance with a totally negative neurologic examination. Eyeground changes need not accompany moderate forms of chronic intracranial hypertension so that the presence of normal fundi is not necessarily an excuse for omitting a spinal puncture for manometric purposes. Finally, in the scheme of special diagnostic investigations, encephalography takes an important place in selected cases, not as a routine procedure. There should be sufficient indication in the subjective picture and the neurologic examination for air injection. This decision is best left to the judgment of specialists in the field, lest undue and unwarranted discomfort be inflicted on patients. Subarachnoidal adhesions over the cerebral hemispheres, obstruction of cerebrospinal fluid flow in the basal cisterns and ventricular distortion are the diagnostic highlights in encephalography, but here again interpretations and evaluations should be made by persons skilled and experienced in this line of work.

Although it is not within the scope of this paper to describe methods of neurologic examination, it will serve the theme to mention organic stigmata which should deter one from making a diagnosis of posttraumatic neurosis or hysteria. Their absence does not, by any means, absolutely exclude the finer type of disseminated central nervous system lesions, as is proved by the universally known clinical fact that many post-concussion cases show no abnormal neurologic signs. Unequal pupils, either one or both reacting incompletely to light, in the absence of syphilis, should command attention. The same is true of the following phenomena: weakness of one or more extraocular muscles, nystagmus, ptosis of an upper eyelid, weakness of the facial muscles on one side, either of the peripheral or central type (seventh cranial nerve or cortical, respectively), deviation of the tongue when protruded towards the side of paretic perioral muscles, motor exhaustibility of maintained elevation of an upper limb as compared to its fellow, and fatigability of a lower extremity while walking. (A hemiplegia or definite focal paralysis is too obvious a finding, hence I refer to exhaustibility of innervation as an indication of moderate motor system involvement.)

Gross sensory loss on one side of the body is rarely encountered. When the parietal cortex is involved the sensory change is incomplete and affects discriminative and gnostic sensory perception rather than

gross loss of the vital sensations. Inequality of tendon reflexes, marked difference in the abdominal and cremasteric reflexes and perversion of the plantar reflex on one or both sides should be given the evaluation they deserve from the organic standpoint, but the normal wide variation of reflex activity should be kept in mind. The positive Romberg sign is too often incorrectly interpreted since it is readily demonstrated in many neurotic cases. The hysteric, as well as the malingerer, will fall to one side or the other the moment he closes his eyes; therefore, a monosymptomatic positive Romberg is not a reliable indication of intracranial pathology.

The critical physician should have no difficulty in differentiating true dysmetria in the finger-to-nose test from the intentional bizarre excursions of the arm with the finger striking every part of the face but the nose. Where tinnitus aurium and impaired hearing are alleged, the audiometer reading is a more reliable basis for determining percentage of functional loss than tests made with the whispered voice, watch tick and tuning forks. Recourse to the audiometer is advisable when the compensation rating before an Industrial Compensation Board is to be made chiefly on the loss of hearing, because there is much room for exaggeration and falsification in claims of this character.

It may seem superfluous to discuss the methods of differentiating hysterical from peripheral nerve paralyses, but not to one who has seen many instances of hysterical paralysis of an upper limb diagnosed as a tear of the brachial plexus. The attitude of the limb and its component parts, muscle nutrition, reflex activity, electrical reactions and the distribution of the sensory loss hold the solution to the problem. It may be said that an allegedly paralyzed limb is not an organic condition if the motor and sensory loss are segmental, i.e., not in conformity with peripheral nerve patterns, if the muscle volume is preserved throughout with no evidence of focal atrophy, if the electrical reactions show no quantitative or qualitative change with the faradic and galvanic currents, and if the reflex arc is intact. Cerebral palsies offer somewhat more difficulty in their differentiation since the reflexes may be within the limits of normal activity and the electrical reactions may not be those of degeneration. However, other features of the symptom complex are generally tantamount to motor weakness and serve to differentiate the condition from an extracerebral disorder. Yet there are numerous instances of cerebral injuries associated with hysterical paralyses in different parts of the body. This makes the situation all the more interesting and is a test of the clinician's acumen and diagnostic ability.

Functional nervous disorders may follow injuries to any part of the body, but the vast majority of the so-called traumatic neuroses are related to head injuries. Hence more space has been given to the discussion of these. Is traumatic neurosis, therefore, a disorder of the central nervous system arising from pathologic changes in the brain after trauma? Is there anything characteristic about the traumatic neurosis situation differentiating it from psychoneuroses not the outcome of trauma? If so, what is the physiopathogenesis and what are the distinguishing clinical markings; if not, why should the term neurosis be dignified by the specific appellation traumatic? If it is so called simply because the neurosis followed an accident, why not in the case of the other neuroses identify them as marital, sex, economic, etc.? There is considerable controversy in the literature on the subject and much that is vague and unconvincing. There are authorities who hold that diffuse microscopic cerebral changes occur in cerebral concussion and that the resultant functional phenomena may take the form of alterations of personality, lowered capacity for energy output, hypersensitivity to the environment and the classical symptoms of headache and dizziness, a symptom complex often referred to as the post-traumatic constitution. On the other hand, authorita-

tive opinions express a contrary belief, to the effect that the neurotic display is an indication of the individual's inadequate handling, emotionally speaking, of the accident experience and its correlated socio-economic circumstances. An obviously biased point of view not infrequently expressed in the literature is the one insisting that a post-traumatic neurosis is based on the wish to obtain monetary redress or the desire to retire indefinitely from a life of laborious routine even though the compensation award means little more than bare subsistence. To show the fallacy of such a bigoted idea is merely to call attention to neurotic invalids who have been made so apparently by a severe injury under circumstances devoid of compensation or litigation values. Undoubtedly there is relative merit up to a certain degree in each theory, but its latitude is delimited by the individual observer's experience, personal attitudes and bias.

When we compare the complete recovery of some patients who have suffered severe brain injuries with the lagging convalescence of those who complain bitterly for many months after a scalp wound with doubtful concussion effects, one must be in doubt about the organic basis of the so-called post-traumatic neurosis. I believe, nevertheless, there is a group of cases in which the original head trauma may have been no more than a mild cerebral concussion but in which microscopic perivascular damage has occurred diffusely through the subcortical white matter of the cerebral hemispheres or in the region of the basal ganglia. The routine neurologic examination nevertheless discloses no deviation from the normal. No focal or localizing functional defects can be recognized, but the resultant clinical picture may be one similar to a neurasthenic state.

Headache and dizziness need not be interpreted as evidence of increased intracranial pressure and involvement of the vestibular pathways, respectively. They may be merely evidence of a general state of impaired health such as we see in constitutional neurasthenics who have had no injuries to their central nervous systems. Symptoms resulting from exhaustibility of the special senses, lowered physical endurance, visceral symptoms well known in neurasthenia, insomnia, etc., may be envisaged as an expression of autonomic dystonia brought about by deep-seated microscopic lesions which interfere with the associational mechanisms within the central nervous system which normally subserve the total integration of the general economy. This form of post-concussional disability is not necessarily permanent although convalescence is prolonged and it may take one to three years for complete rehabilitation. I believe this group of cases constitutes a small portion of the general class of traumatic neuroses. I do not think it should be included under that caption, but the differentiation is extremely difficult.

It is my opinion that the most common form of post-concussional disability is caused by a temporary paresis or instability of the intracranial vasomotor mechanism if only for the clinical reason that the majority of such patients are clinically short-lived. Progressive spontaneous amelioration is the rule providing no adverse emotional factors are brought into play. It is in the protracted case that one attempts to differentiate between the type envisaged in the paragraph above and the psychoneurotic disorder; at this time Kennedy's statement (see above) is pertinent. A clear cut exception to this dictum may be noted in the form of unilateral tinnitus aurium, impaired hearing, unilateral pain in the head and postural dizziness following a sharp blow to the temporoparietal region even without loss of consciousness. The annoyance created by the unremitting character of these symptoms may provoke an agitated frame of mind in certain individuals and thus give the symptom complex an hysterical coloring. The lesion is probably a labyrinthine concussion, with or without hemorrhage, and need not necessarily be associated with much of a concussion effect on the brain. The emo-

tional instability of the patient may increase as time goes on and the original symptoms continue without improvement. Therefore, the persistence of symptoms in this type of case for longer than four months would not point to the neurotic nature of the disorder just because the criteria of brain injury are lacking.

If the term traumatic neurosis were to be used in a refined form it could be said that there is only one true traumatic neurosis, namely, the Schreckneurose of Horn (terror neurosis, as translated by Strauss and Savitsky). It implies that the etiology of the condition is to be found not in the physical effects of the injuries, but in the terror which accompanies the accident; it occurs under such circumstances that the patient has time to be aware of and be terrorized by the impending disaster with a serious threat to life. According to Ebaugh and Benjamin the syndrome is frequent in war and earthquakes, occasionally in mining catastrophes and sea disasters, but almost never in the ordinary industrial and automobile accidents. They further state that it has seldom followed severe head injuries because the loss of consciousness and retrograde amnesia protects the patients to some extent from the psychological sequelae of terror. The symptoms are usually those of a marked anxiety disturbance with frequent dreams in which the patient relives the traumatic situation. The natural personality make-up of such an individual need not necessarily have been neurotic. The traumatic situation as such may be considered a true causative factor in contradistinction to all other neuroses following trauma. The condition tends to clear up spontaneously after a varying length of time, providing no additional psychoneurotic or hysterical factors intervene, such as might be created by compensation or litigation interests with their attendant mental and emotional strain. It stands to reason that a terror neurosis would play greater havoc in an individual with preëxisting mental or emotional instability and the prognosis would thus become proportionately less favorable.

The conscious desire to receive compensation and indemnification for injuries sustained in industrial and other types of accidents unquestionably plays a prominent rôle in influencing post-traumatic syndromes especially of the neurotic type. The influence may be expressed in terms of secondary psychogenic symptoms, exaggeration of residual organic distress and malingering; the combination of these phenomena makes rational the use of the term hystero-malingering. Complicated medicolegal situations frequently develop from these human elements and results in much confusion of thought and expression of opinion before Workmen's Compensation Boards and in courts. It is an unfortunate phase of modern human experience for which as yet there is no solution. There is no set of diagnostic criteria which can be clearly set forth to aid one in separating the wheat from the chaff, so to speak, because each case must be considered on its own merits. Every aspect of the particular problem should be critically surveyed. The physician must first be certain that his view is not prejudiced by preconceived ideas and attitudes in regard to the so-called compensation or litigation complex of the patient in question. The oft-times all too obvious behavior of a litigant may be misconstrued as an indication of malingering in spite of coexistent bona fide traumatic residuals, when, as a matter of fact, the overt behavior may be emotionally colored by a natural defense attitude towards forces operating against the personal interests of the individual. Anxiety, apprehension, emotional unrest and exaggeration of residual traumatic symptoms become grouped together in a symptom complex which may be referred to as a compensation or litigation neurosis. An unjustifiable conclusion is that a compensation or litigation neurosis is synonymous with frank malingering. I must admit, however, that many cases of this type require hair-splitting analysis before all interested

parties are convinced. It is here that we find the largest bone of contention in medicolegal evaluations and adjudications.

Malingering without neurosis is not so common a human fault as one might be led to believe by the character of medical testimony. It is encountered in liability cases more often than in industrial accidents because the stakes are higher. In a large experience a physician will occasionally meet with a clear-cut picture of protracted malingering following an accident but he should have no difficulty in recognizing it. The unadulterated malingerer, i.e., without a psychoneurotic background, must be caught at his own game if the physician is called upon to identify the disability for legal evaluation, and the individual in question is proved to be a malingerer in direct proportion to evidence of his physical capabilities contrariwise to his claims. There are numerous tests for malingering as it may affect the special senses or general somatic functions, but the ingenuity of the physician will generally suggest specific methods for the individual case and he will invariably be able to pick out the flaws. Hystero-malingering is a much more frequent problem in which the psychogenic situations are paramount in value to the intentionally exaggerated or maligned features of the clinical picture. It is the belief of some authorities that malingering is an expression of psychopathy in one form or another, but in my opinion, the psychopathology of malingering per se should afford the culprit no respite from responsibility, whereas, exaggeration by the hysterically disturbed individual is relatively excusable.

The measure of success in the treatment of post-traumatic functional nervous disorders is to be found in the personal relationship and rapport between the physician and patient, but not solely in the sympathetic atmosphere created because well-meaning but injudicious remarks regarding diagnosis and prognosis may have adverse effects. Not infrequently much of the responsibility for protracted invalidism can be laid at the feet of the first medical attendant who solicitously commented on the very narrow escape the patient had, or discussed details of brain pathology and suggested an extended period of convalescence with the possibility of permanent residuals. In other words, the less said in the presence of one fresh from the experience of an accident situation the better. If it is necessary to do a diagnostic spinal puncture in a head injury it is unwise to acquaint the patient, if he be conscious or at a later date, with cerebrospinal fluid hydrodynamics lest it fix in his mind the idea of brain pressure and expanding mechanisms resulting in severe headache. A deplorable attitude on the part of some physicians towards medicolegal cases is the different approach from that in traumatic cases of no litigation value. In the latter instance he will reassure the patient as to the benignity of the injuries and the excellent prognosis and will encourage resumption of normal activities in line with physiologic recovery; in the other type of case he feels that it is necessary to have a period of bed confinement to establish the gravity of the primary reactions and later cautions the patient at every step lest he hamper the doctor's therapeutic efforts thus establishing himself as an indispensable part of the medicolegal situation with its ultimate satisfying monetary returns. The patient is thus unwittingly nurtured in his incipient neurosis, and not infrequently, I am sorry to say, aided and abetted in his litiginous prosecution. The occasional physician of this type is not the chief offender in molding the psychogenic post-traumatic picture, for we owe much to the strong suggestive influence of the attitude of relatives, friends and counsellors-at-law.

Psychotherapy is the mainstay in the management of the traumatic neurosis patient. Explanation, reassurance, persuasion and suggestion are the dynamic factors of the method. Reassuring explanation of symptomatic distress can be acceptable only if the physician is fully conversant

with the factual data of the accident and the sum total of the resulting injuries; therefore, a detailed history should be elicited. Nothing is more reassuring than an apparently painstaking physical examination, the word "apparently" being used to suggest more patience with details which might impress the patient with the thoroughness of the examination. The next move is to efface morbid conceptions of organic pathology which may be held by the patient. This naturally follows on the heels of the complete anamnesis and the thorough examination.

Then the attack on individual symptoms is planned, with physiotherapy in its various modalities judiciously and tactfully applied to the various distressed areas. The value of physiotherapy is in direct proportion to the psychotherapy meted out with it. This stage of the program is the longest and most variable; the physician's patience may be tried many times by refractory reactions of the patient, but it is at these vital psychological points that the physician must drive harder with his psychotherapeutic armamentarium, namely, explanation, reassurance, persuasion and suggestion.

An attempt must be made to bring about a wholesome attitude towards the compensation situation or litigation possibilities in the individual case, most often an insurmountable obstacle, but rehabilitation cannot be complete without this accomplishment. Serious deterrents are often encountered in the form of unemployment facing the claimant after recovery, a preference for an extended period of inactivity on compensation rather than return to laborious work, utilization of the accident situation as an avenue of escape from pretraumatic situations such as personal illness or infirmities or family conflicts, etc. In liability cases pending trial it is often impossible to influence the clinical course until final settlement of the legal matter is effected. A quality well worth cultivating in one's self is the conscientious attempt to dissociate the legal from the medical values in the manner of the approach to the patient at every point of the way with the hope that the patient will be similarly influenced by example. A conscientious prognosis cannot be given without this attitude on the part of the physician, and nothing is more potent therapeutically than a consistently optimistic prognosis.

Symptomatic treatment need be given little discussion since there is nothing specific in its nature, but a word of caution is given against the indiscriminate and prolonged use of bromides and barbiturates; effective therapeutic doses of these drugs, given only when really indicated rather than as a panacea for nervousness, will do all that is desired. Prolonged bed cures for post-traumatic hysterics are to be discouraged, but conservation of physical and mental energies should be supervised.

SUMMARY

Trauma to the psyche and soma is often followed by prolonged functional disabilities out of all proportion to the severity of the injuries suffered, and since this is by far more true in cases where compensation and litigation are involved, the post-traumatic syndromes take on greater socio-economic significance.

In cases of head injury the post-traumatic triad of headache, dizziness and nervousness constitutes a veritable battleground for medicolegal opponents with expressions of opinion varying from malingering to widespread petechial hemorrhages in the brain.

The chronic back invalid is figuratively roasted with never-ending local baking when the physiopathogenesis may either be psychogenic or occasionally an unsuspected dislocation of the nucleus pulposus. In the former instance local tissue pathology is envisaged and in the latter case a psychoneurosis pictured with local therapy as a psychotherapeutic aid.

The gamut of neurotic and hysterical phenomena is enumerated with an attempt to evaluate them in the total setting as

cases of post-concussion syndrome, traumatic neurosis or hysteria, compensation or litigation neurosis and malingering. Not infrequently fragments of each group are combined in a setting deserving the term of hystero-malingering.

It is pointed out that after a reasonable period of convalescence (Kennedy, four months) a history of head injury does not always suffice to identify unremitting symptoms as exclusively post-concussional. Personality factors, social and economic data as well as the legal values involved must come in for close scrutiny as potential psychogenic factors in prolonging the alleged posttraumatic disabilities.

Although psychotherapy is the dominant note in the handling of the post-traumatic functional disorders, a thoroughgoing clinical investigation for possible persistent organic defects must precede its institution. Explanation, suggestion and persuasion when meted out with patience and tolerance constitutes the reëducational program of psychotherapy which leads to rationalization and ultimate rehabilitation.

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ROENTGENOLOGIC CONSIDERATIONS OF CERTAIN JOINT INJURIES

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WHEN Roentgen discovered the *x*-ray, he brought to medicine a method of diagnosis which has helped in solving many problems, not the least difficult of which are fractures occurring in and about the joints. Here, the surgeon's skill and ingenuity are taxed to the utmost in order to obtain a satisfactory functional result.

FIG. 1. Normal ankle, showing weight-bearing line.

The diagnosis of joint injuries is incomplete without a thorough *x*-ray examination before reduction; later treatment should be verified by numerous check-up films after reduction. Too few films are made in many cases, resulting in a disappointed patient and perhaps in an eventual law suit. In many instances the use of the *x*-ray determines either the question of disability or of a good functional result.

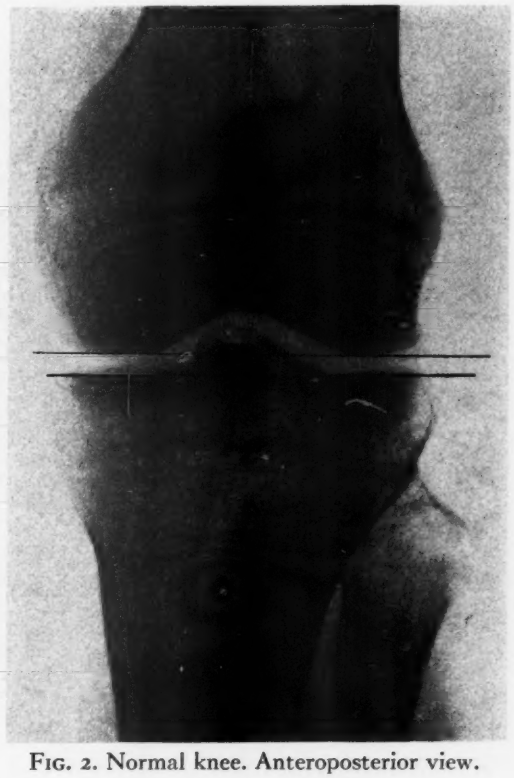

FIG. 2. Normal knee. Anteroposterior view.

Observations of the normal articulations show that the various bones have certain definite anatomic relationships and that if these are disturbed, a good functional result cannot be expected. Even in cases where there have been severe injuries to the soft parts, nature has a way of healing these so that if accurate reposition of displaced fragments is accomplished, one will have a satisfied patient in the majority of instances. Functional results may be predicted in the ankle, knee, hip, elbow and wrist, also in lumbosacral and upper cervical injuries, if one will make certain linear measurements on the *x*-ray film.

ANKLE

Ankle joint injuries are common, usually being due to the application of direct force applied in various directions. As the ankle is a hinge-joint, being held in position by ligaments, any force applied in an abnormal direction results in a sprain, dislocation, or fracture, or a combination of these. If one examines an anteroposterior *x*-ray film of a normal ankle, one will see that if a line is projected through the middle of the tibia, extending into the astragalus, this forms the weight-bearing line and bisects the superior articular surface of the astragalus. (Fig. 1.)

Fig. 3. Normal hip. Shenton's line.

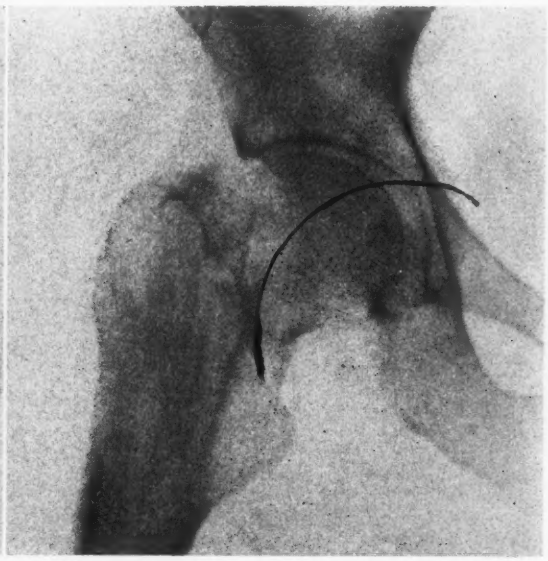

Fig. 4. Fracture of the neck of the femur.

After reduction of a Potts' fracture, with or without dislocation, if the weight-bearing line does not bisect the astragalus, look for an unsatisfactory result and a permanent disability. In some fibular fractures, there may be a slight deviation of the distal fragment. However, if the ankle mortise and weight-bearing line are undisturbed, a satisfactory function should be obtained. Malleolar fractures demand the same care in reduction as Potts' fractures. Incidentally, the term Potts' fracture is unsatisfactory, classification of ankle injuries should be made according to the portions of the bony structure involved.

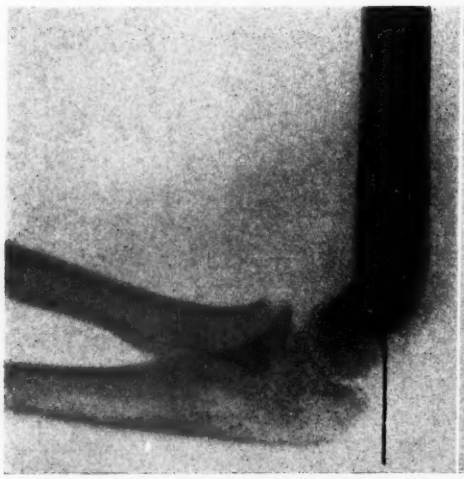

Fig. 5. Normal elbow. Lateral view.

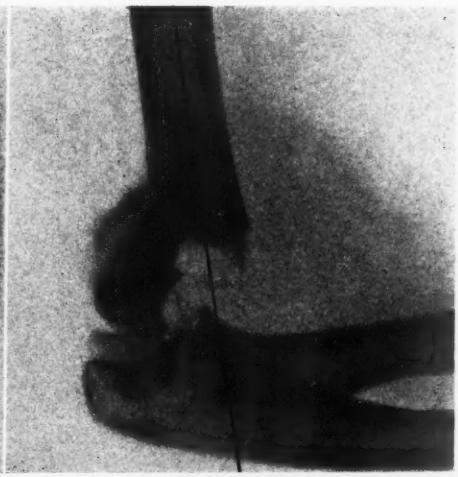

Fig. 6. Supracondylar fracture of humerus.

KNEE

The knee joint is subjected to many injuries which often result in serious disability. In the so-called "bumper" accidents, the proximal portion of the tibia is most often involved. An anteroposterior *x*-ray film of a normal adult knee shows that the distal portion of the femur articulates with the proximal portion of the tibia, there being some separation of the bones indicating the joint space. If one is confronted with a fracture of the outer tuberosity of the tibia, the joint level between the outer and inner tuberosities will be unequal. The amount of depression can be measured by drawing a line on the film through the tibia in a longitudinal direction, and another line at right angles to this first line at the level of the uninjured tuberosity. Measurement with a straight edge will at once determine the amount of the depression of the injured structures.

Another measurement of value is to draw a line across the proximal surfaces of the tibia at the joint level and another line between the articular portions of the external and internal condyles of the femur. Normally, these lines will appear practically parallel. (Fig. 2.)

HIP

The normal hip joint shows that the head of the femur articulates with the pelvis at the acetabulum. Many years ago, Shenton showed us "Shenton's line," the means whereby we could predetermine correct anatomic position. In the normal case, a line is drawn on the anteroposterior *x*-ray film along the medial curved portion of the neck of the femur and is continued along the inferior border of the ascending ramus of the pubis. This line forms an unbroken curve. (Fig. 3.)

Now suppose that we have a fracture of the neck of the femur. (Fig. 4.) If a projection of the above line is made along the neck of the femur and continued toward the median line of the body, it will be seen that it does not coincide with the line above described. Reduction is accomplished when this line approaches the normal "Shenton's line."

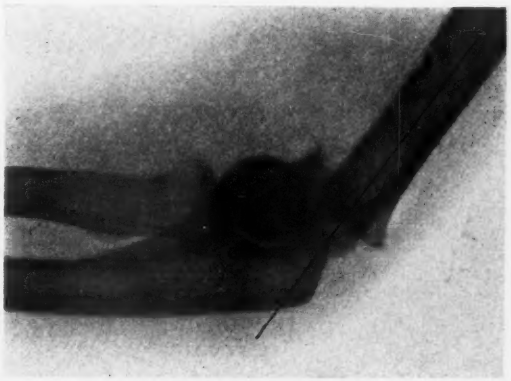

Fig. 7. Reverse supracondylar fracture of humerus.

In the lateral projection on the *x*-ray film, displacement must also be considered. In these days when nailing of hip fractures is being done, more accurate diagnoses must be made. All hip fractures require anteroposterior and lateral films.

ELBOW

One of the most crippling fractures in children, if a good reduction is not obtained, is the supracondylar fracture of the humerus, caused by falling on the outstretched or extended hand. Many of these fractures are clinically diagnosed erroneously as dislocations. While dislocation does occur, the greater percentage of the injuries are fractures.

Ashhurst has ably described these fractures and has shown us how to obtain satisfactory results. The *x*-ray film, however, will tell us how to predict good functional results, provided there is no nerve injury. A lateral film of the elbow shows us in the normal that the distal portion of the humerus curves a trifle forward so that if a line is drawn longitudinally through the humerus, the articular surface will be anterior to this line. (Fig. 5.) In a supracondylar fracture the articular portion will be found posterior. (Fig. 6.) The *x*-ray film must be so made that there is no rotation of the forearm. In a reverse supracondylar fracture, the distal portion of the humerus

will be displaced forward a great deal more than is normal and the normal line will pass posterior to the articular portion of the humerus. (Fig. 7.) Accurate reposition of the fragments of the humerus must always be accomplished unless one is to be confronted with a disabled elbow. Here again, anteroposterior and lateral films are essential for diagnosis.

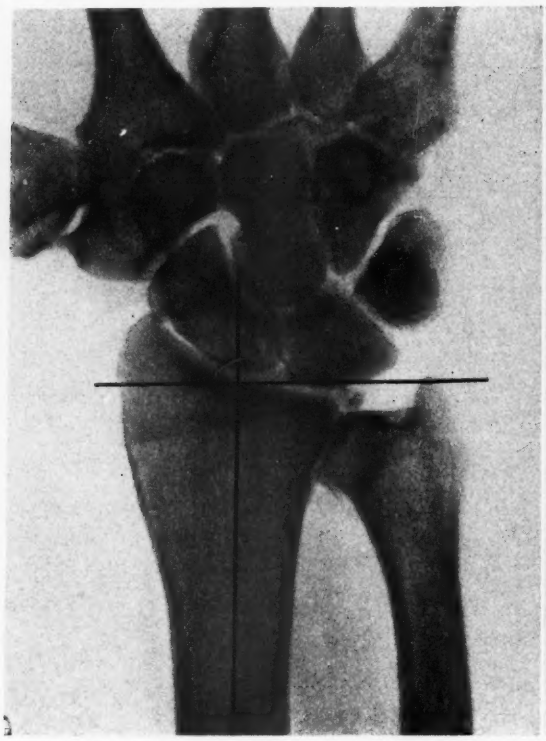

Fig. 8. Normal wrist. Anteroposterior view.

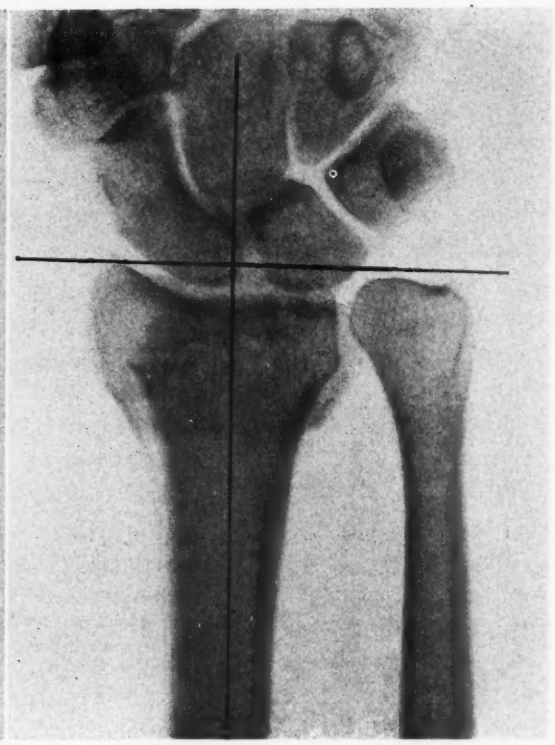

Fig. 9. Colles' fracture. Anteroposterior view.

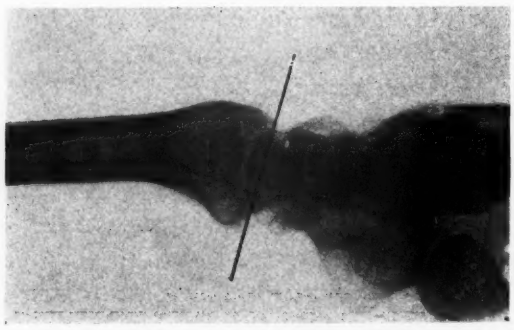

Fig. 10. Normal wrist. Lateral view.

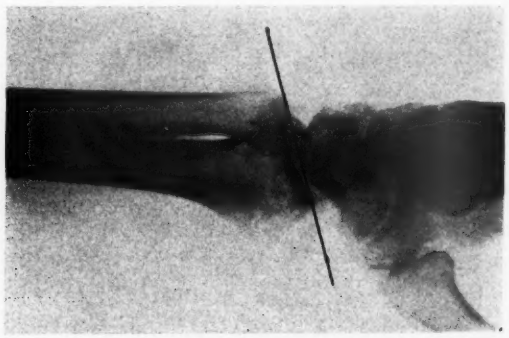

Fig. 11. Colles'. Lateral view.

WRIST

Colles' fractures have been reviewed and described in the literature to such a large extent that it hardly seems necessary to call attention to them in this discussion, but in spite of this, poor functional results are still with us. These are unwarranted in a large majority of cases and if surgeons would remember that there is a method of predetermining whether or not a good result will be obtained, better looking wrists and more useful ones would turn the wheels of industry.

When we examine an anteroposterior view of a normal wrist, we see that if a line is projected lengthwise through the middle

of the radius and into the wrist joint, and another line projected at right angles to this line from the tip of the styloid of the

Fig. 12. Normal lateral lumbosacral.

ulna, the styloid of the radius will be distal to the second projected line. (Fig. 8.) In a Colles' fracture, the styloid process of the radius will be proximal to the second line. (Fig. 9.) This means that an unsatisfactory result will be obtained if correction of the displacement is not accomplished.

Both lateral and anteroposterior views of the wrist are always necessary to determine the position of the broken fragments. If, after reduction, the joint surface of the radius does not point forward and anteriorly, an unsatisfactory wrist will be the result.

The lateral *x*-ray film of a normal wrist shows that if a line is projected between the superior and inferior articular portions of the radius, it will be tilted forward. (Fig. 10.) In a Colles' fracture, it will be tilted backward, forming the typical silver fork deformity. (Fig. 11.) The use of the above lines will predetermine whether or not reduction has been accomplished.

SPINE

The industrial surgeon is called upon many times to decide whether or not

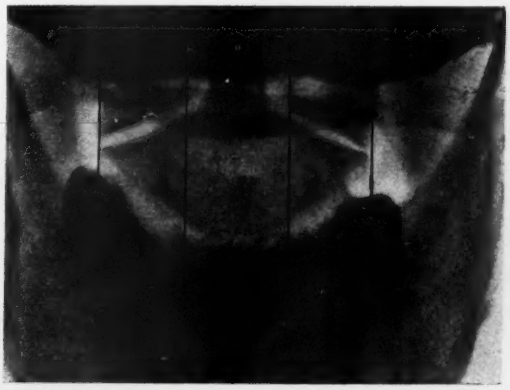

Fig. 13. Normal upper cervical vertebrae. Anteroposterior view.

spondylolisthesis is present. As there are a number of degrees of displacement, more than a casual glance at the film is necessary to reach a decision as to the extent of dislocation. In the lateral lumbosacral *x*-ray film, a line should be projected along the superior portion of the sacrum, and in the area in which the anterior articular border of the sacrum appears, another line should be projected at right angles to the first, which will just cut the inferior anterior portion of the fifth lumbar vertebra. (Fig. 12.) If a third or fourth degree dislocation is present, it will be apparent at once that no measurements are necessary, but in a first or second degree dislocation, the eye will sometimes deceive us so that measurements should always be made.

Stereoscopic films of the lumbosacral area in the anteroposterior direction are often misleading and if the technician has not been careful to center the central ray in the right spot, confusion results. As a matter of fact, one should put his faith in measurements on the lateral film more often than in the stereoscopic anteroposterior.

Injuries to the upper cervical spine are relatively frequent and as these are of a serious nature, definite measurements on the *x*-ray film must be made. The technique of *x*-raying the upper cervical vertebrae is not particularly difficult, but strict atten-

tion must be paid to proper centering of the central ray. We are concerned here only with the axis and atlas measurements, showing whether or not correct anatomic alignment is present.

Fig. 14. Normal lateral upper cervical area.

Fig. 15. Injury to atlas and azis.

If we look at an anteroposterior film of the cervical area, we should see portions of the atlas, the lateral masses, the intervertebral space and the body of the axis, and sometimes the occipital bone if sufficient angulation is present. In order to determine the normal, lines are drawn from the superior medial articular processes of the axis to the median border of the atlas, also from the lateral portions of the atlas to the superior lateral articulations of the axis. All of these lines should be essentially parallel. (Fig. 13.)

On the lateral film of the upper cervical vertebrae, one draws a line from the anterior portion of the body of the axis to the anterior portion of the tubercle of the atlas, another line being drawn from the posterior surface of the body of the axis through the posterior portion of the odontoid process. These lines should also be approximately parallel. (Fig. 14.)

In Figure 15, it will be seen that neither line can be made to appear in its normal position, therefore bony injury must be present.

CONCLUSION

In this paper, an attempt has been made to review some of the more common joint injuries, showing how it is possible to predict functional results by means of linear measurements on the *x*-ray film. Progress to date along these lines, has been made possible by anatomists, surgeons and Roentgen-ray specialists and there is no thought on my part of presenting anything new. As Mademoiselle Bertin has said, "There is nothing new except what is forgotten."

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X-RAY EXAMINATION OF CLAVICLES AND ACROMIOCLAVICULAR JOINTS

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THE anatomic arrangement of the shoulder girdle, with the sternal attachments and the marked curvature and shape of the clavicle, makes it a difficult structure to study in various angles. In fact, some works on roentgenographic technique[1] advise only one view, leaving further study to clinical examination. This is a most unsatisfactory method where a medicolegal angle enters and it is desirable to have as complete a visualization of the involved structures as possible. The diagnosis of questionable fractures, various degrees of actual and alleged acromioclavicular joint pathology and the appraisal examination of end results in fractures of the clavicle all require the best *x*-ray visualization.

Quesada,[2] in March 1926, described an excellent technique for obtaining views of the clavicles in right angle planes. These views should be taken in postero-anterior view only. This technique can be used with the patient in the vertical or standing position, providing one has ample range of position of the tube.

We have frequently observed the clearly visualized clavicles on chest plates taken at 72 inches. In July of 1937 we found it advantageous to prepare films in antero-posterior view at 72 inch distance with the patient standing in a vertical position. Later a posterior-anterior view was added in special cases; other variations by raising or lowering the tube given additional angles of study if desired.

Seven by 17 inch films with double screens in cassettes with 65 to 70 kilovolts at 50 milliamperes for 0.4 to 0.5 (½) second exposure at 72 inches has been a satisfactory technique. The patient holds his breath.

Some of the advantages of this technique have been found to be:

1. Undisputable control study of the opposite, unaffected clavicles, acromioclavicular joint or even shoulder. The fact that both clavicles appear on the same film as the result of a single exposure in a symmetrical position on the film makes it very simple and convincing to demonstrate pathology of this region to laymen, such as arbitrators, commissioners, claim men and jurors.

2. It gives an excellent *x*-ray study of the clavicle, both sternal and acromial ends, as well as full length of the body of the clavicle. The coracoid process and acromion are readily visualized.

3. These films are taken with the patient in the standing position, which is the natural position for functional study of the shoulder girdle and especially advantageous in the study of acromioclavicular dislocation and separations. Most acromioclavicular separations fall back into normal position when the patient is placed in a horizontal position and may give an erroneous *x*-ray study.

4. There are almost as many variations of size, shape, posture and articulations of the clavicle as there are variations of features and for this reason the normal for the individual to be studied is very desirable. Some clavicles are quite straight in certain views, some curved, some drooping and in others the sternal ends are carried high. In the view recommended we set up a normal study of the clavicle of the person to be examined.

5. In the appraisal of the end result in fracture of the clavicle these films are particularly advantageous. Taken at 72 inches, the minimum distortion occurs and by being taken in symmetrical position the actual measured length of the fractured clavicle can be compared to the normal clavicle on same film. The sternal and acromial ends are both plainly visualized and the length of the clavicle easily measured.

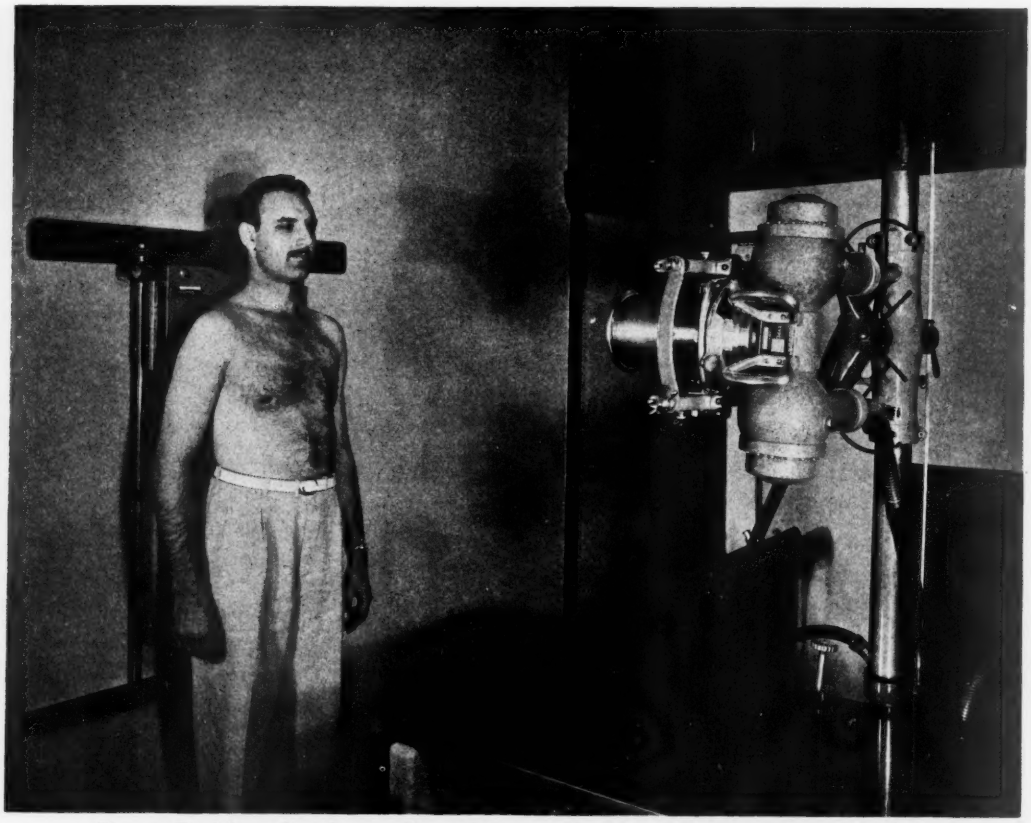

Fig. 1. Position of patient, tube and cassette in anteroposterior view of clavicle.

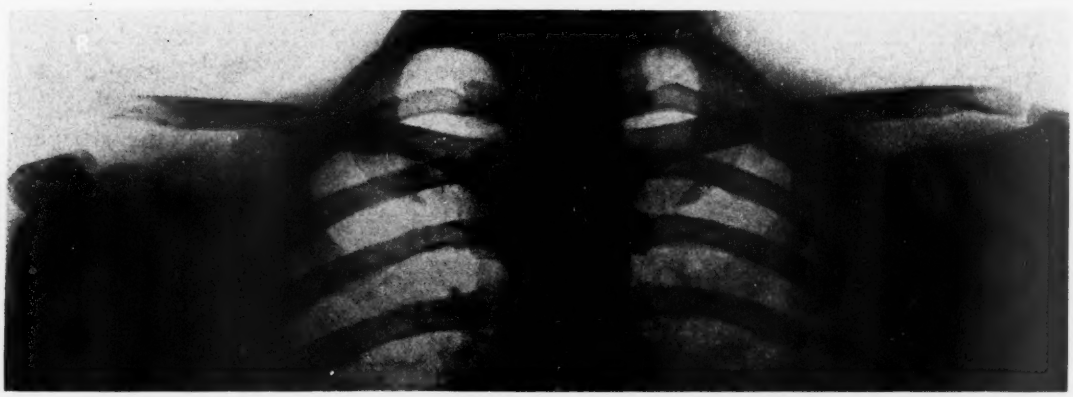

Fig. 2. A single view, in an *x*-ray film 7 × 17 inches, visualizes the full length of both right and left clavicles with acromioclavicular joint separation on the right. Sternal ends of clavicle symmetrical. (R indicates right side.)

6. In nearly all women and in most men the 7 × 17 inch film is long enough to visualize fully not only both clavicles but also both shoulder joints, which can likewise be studied on this one view. Only in

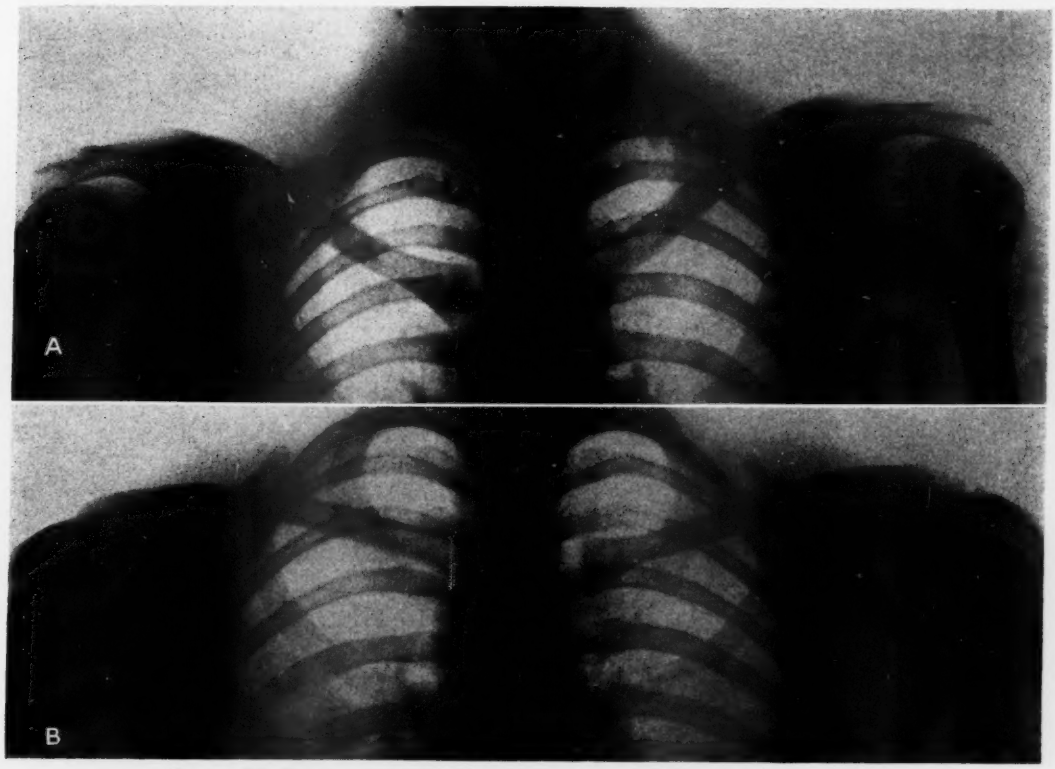

Fig. 3. W. R. A, anteroposterior normal. B, postero-anterior normal.

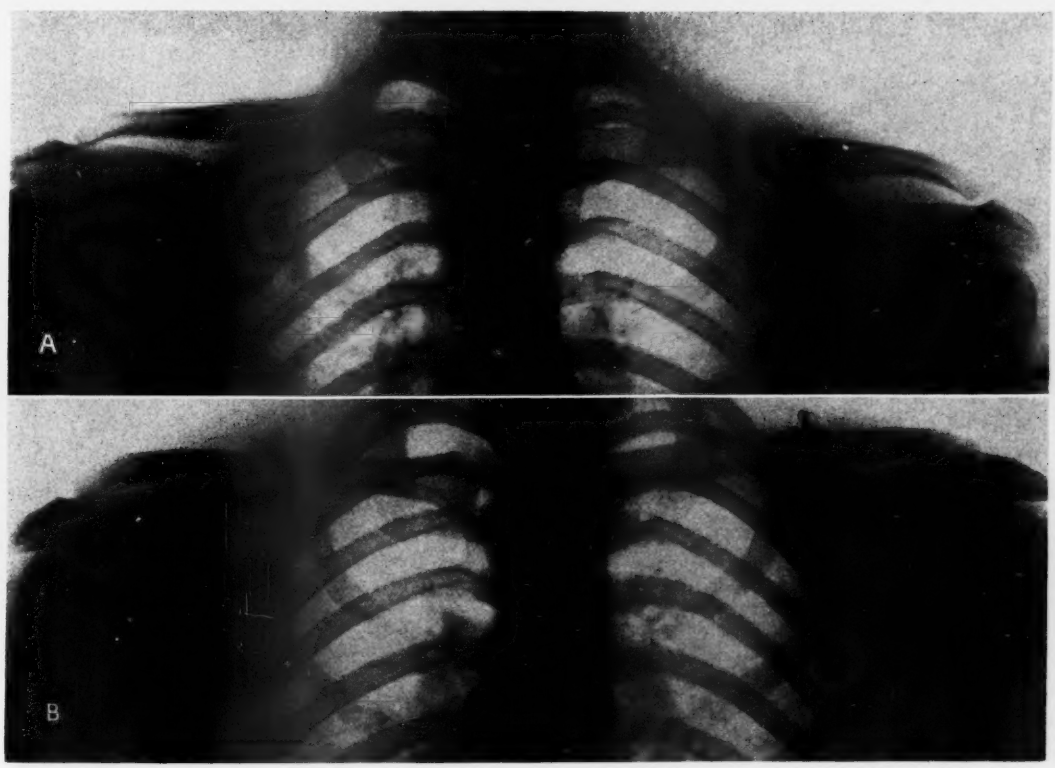

Fig. 4. M. K. A, anteroposterior. B, postero-anterior. Normal variations, almost straight clavicles with sternal ends elevated. Clavicles almost at right angles to spine. (Compare with Fig. 3.)

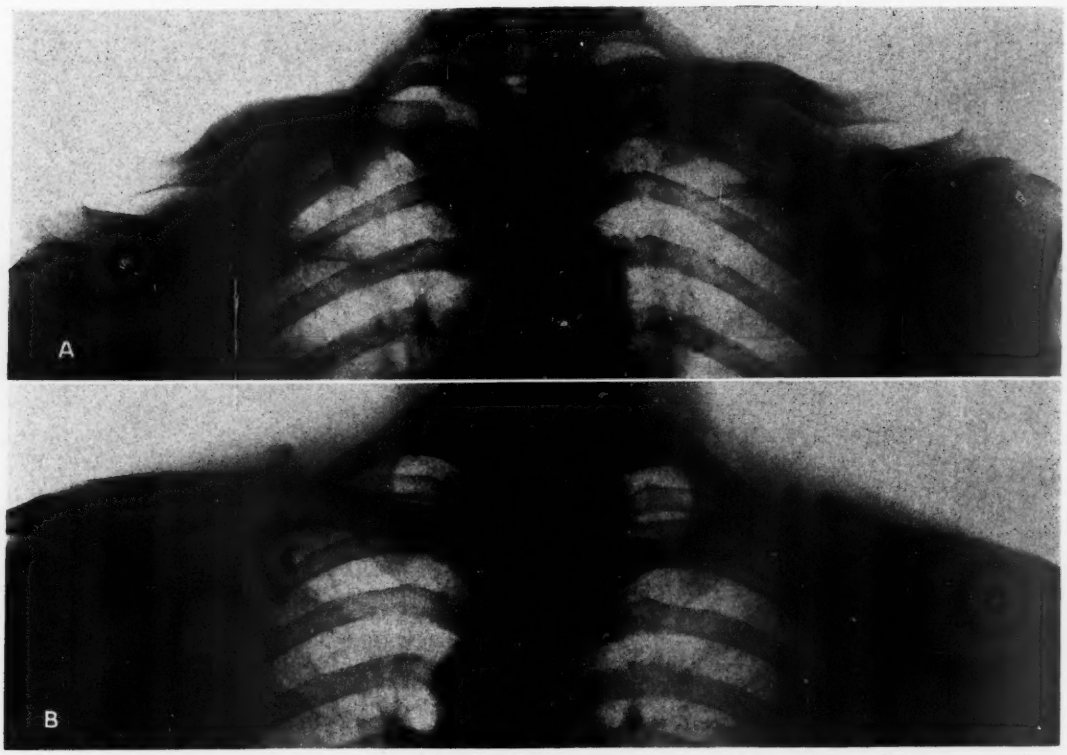

Fig. 5. C. T. A, anteroposterior view with tube lowered. Line of ray directed from below upward. B, postero-anterior, line of ray horizontal—at right angles to body. Normal variations.

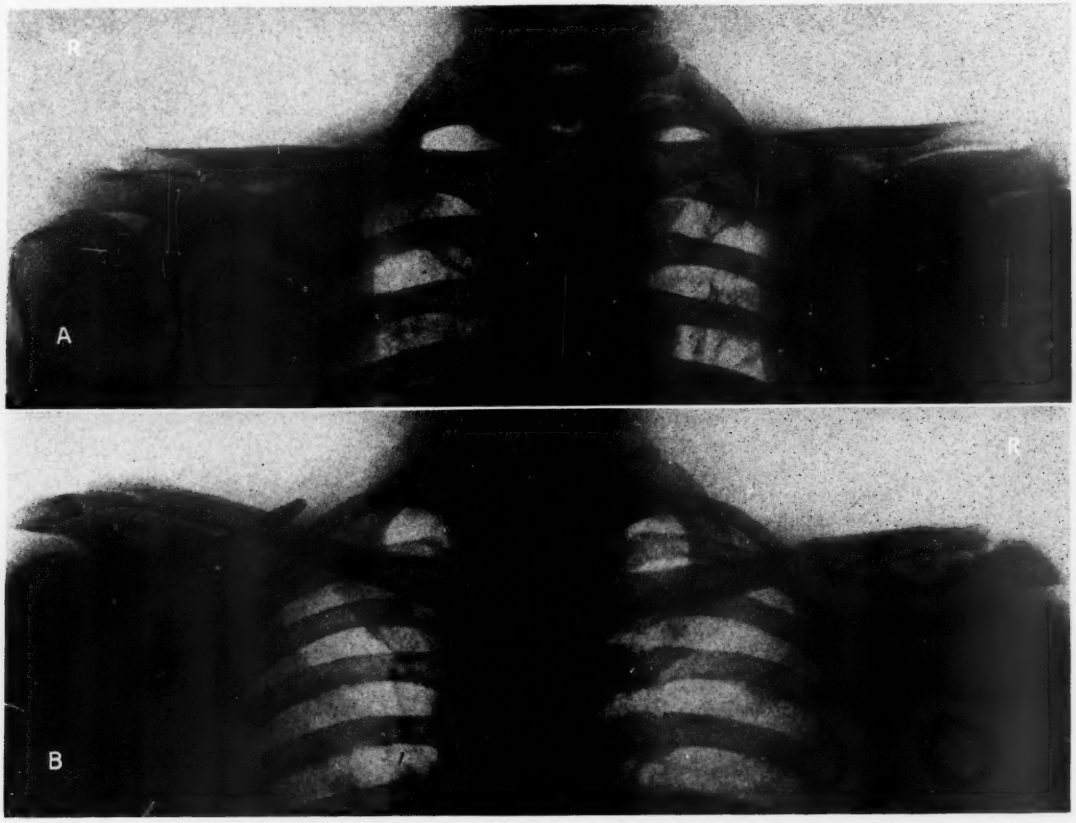

Fig. 6. F. T. Early arthritis. Acromioclavicular joint. A, anteroposterior view; note sharp demarcations and joint margin on left, and hazy irregular roughened right, with some lipping and early exostosis. B, postero-anterior view—good visualization of sternal ends of clavicles. Full lengths of clavicles can be measured and compared with ease. (R indicates right side.)

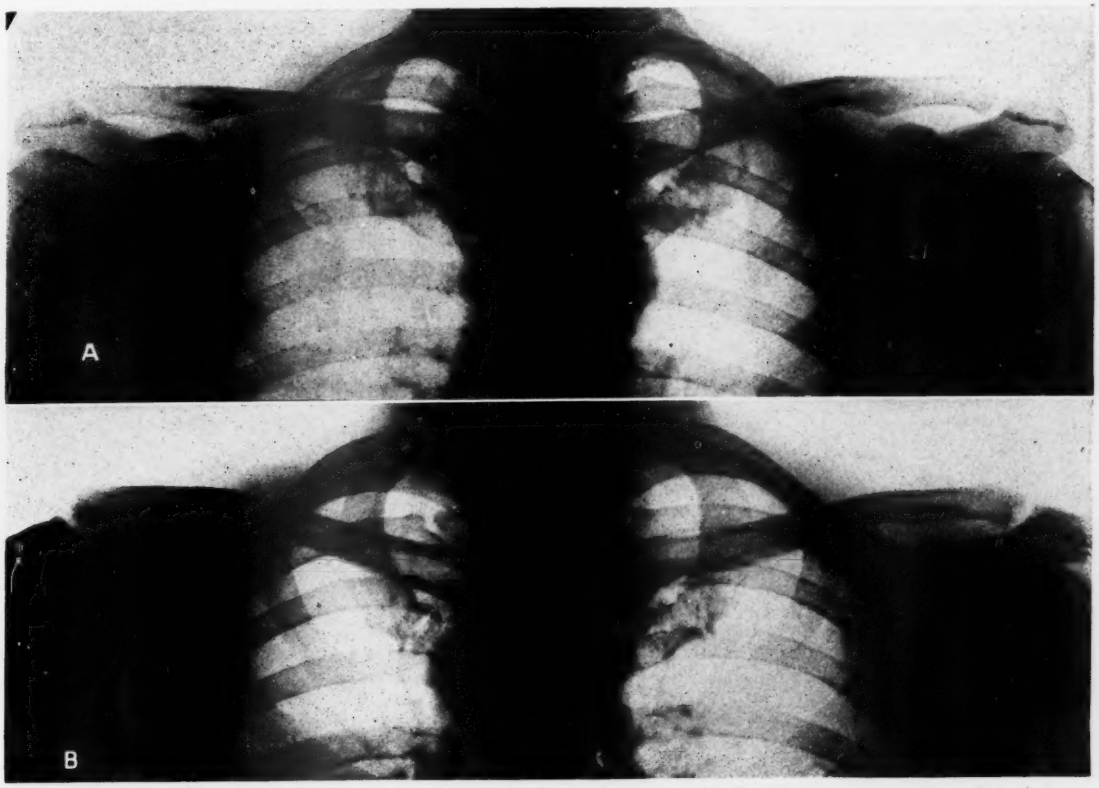

Fig. 7. J. O. K. More advanced arthritis. Acromioclavicular joints both sides. A, anteroposterior view. B, postero-anterior view. Joint spaces diminished. Joint surfaces irregular, with lipping and osteoarthritic deposits.

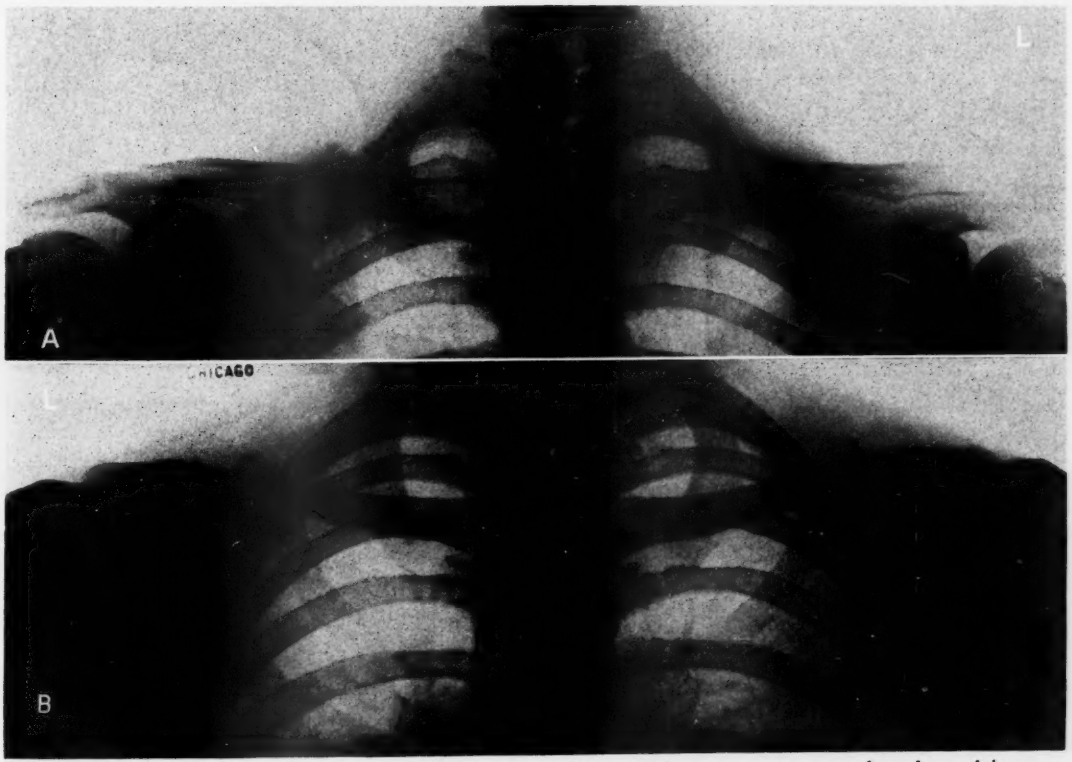

Fig. 8. A. S. Slight acromioclavicular displacement and separation. A, anteroposterior view with arms parallel to sides. Shows displacement on left. B, postero-anterior view with arms backward, hands on lumbar region. Displacement not visualized. (L indicates left side.)

persons with especially broad shoulders do the shoulders extend beyond the margins of the films.

found it advantageous to place the marker so as to label the affected and involved side with the letter R or L.

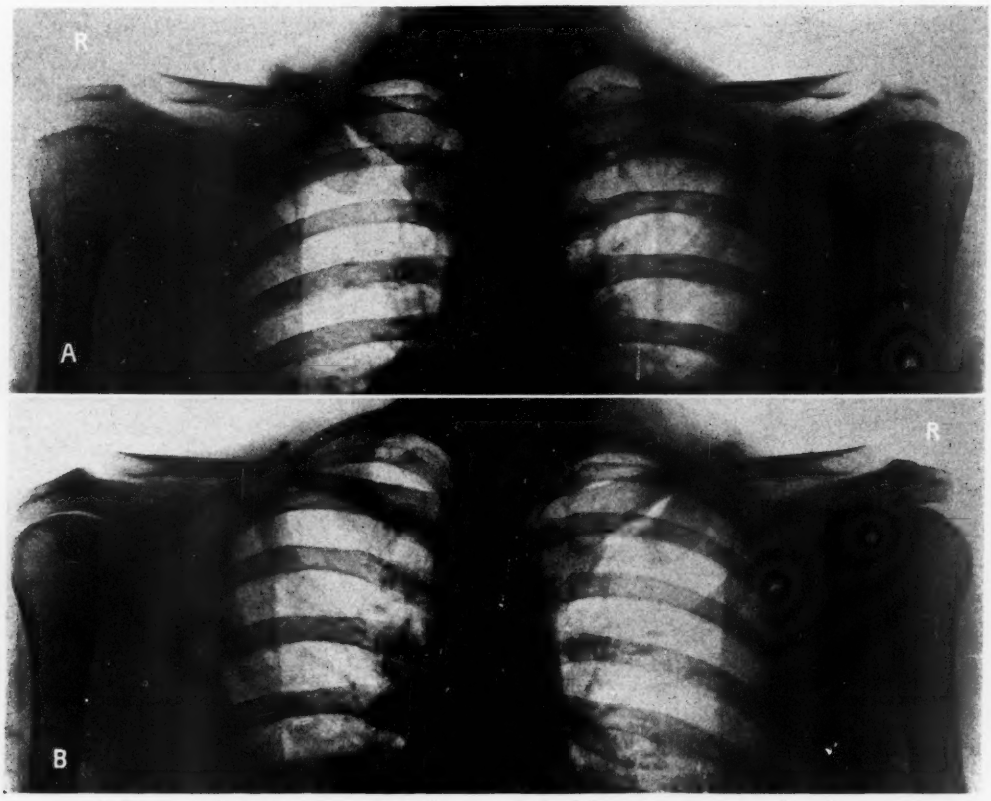

FIG. 9. M. S. Appraisal of end result in fractured clavicle. A, anteroposterior view. B, postero-anterior view. Overriding angulation and shortening of right clavicle visualized. (R indicates right side.)

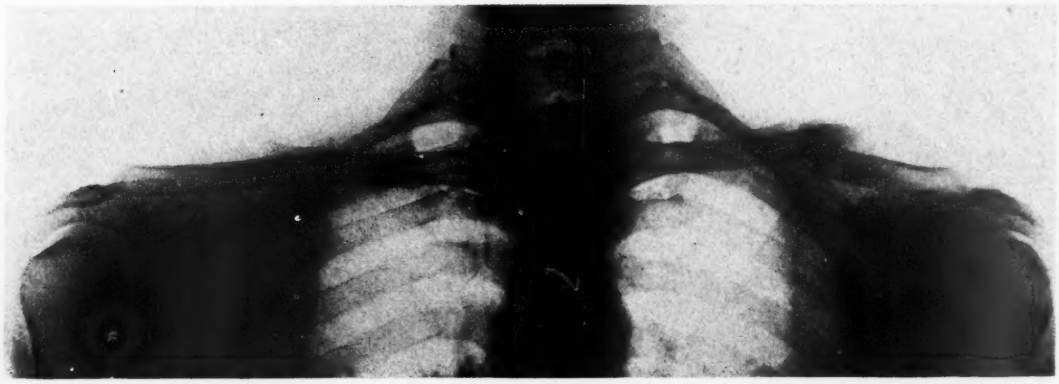

FIG. 10. G. D. F. Fractured clavicle. Delayed union. Appraisal of end result. Fracture of middle third, overriding slight angulation and ball-like callous union.

7. By placing the marker in such a position that it always faces the tube, one has definite information that the particular view being studied was taken antero-posteriorly or postero-anteriorly. We have

8. Economy of films and exposures. In this study nearly always a single 7 × 17 inch film is all that is needed. This gives the maximum amount of information with the minimum amount of

tube exposure and film exposure and also saves the time of the technician and the patient.

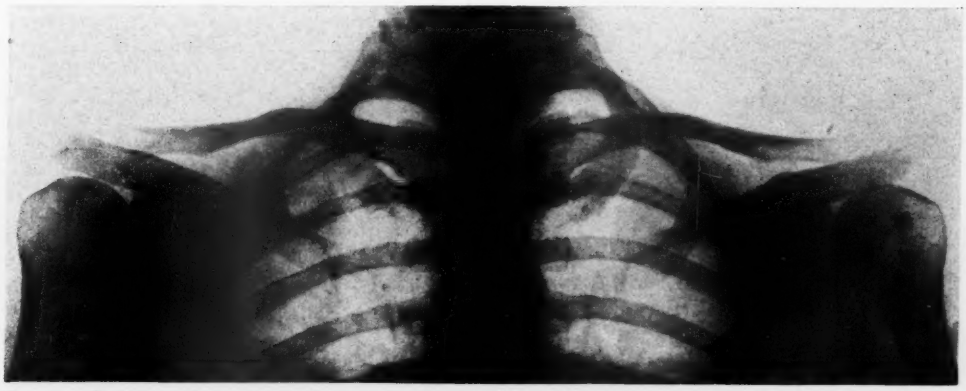

Fig. 11. R. D. V. Both shoulders as well as clavicles visualized on a single 7 × 17 inch film.

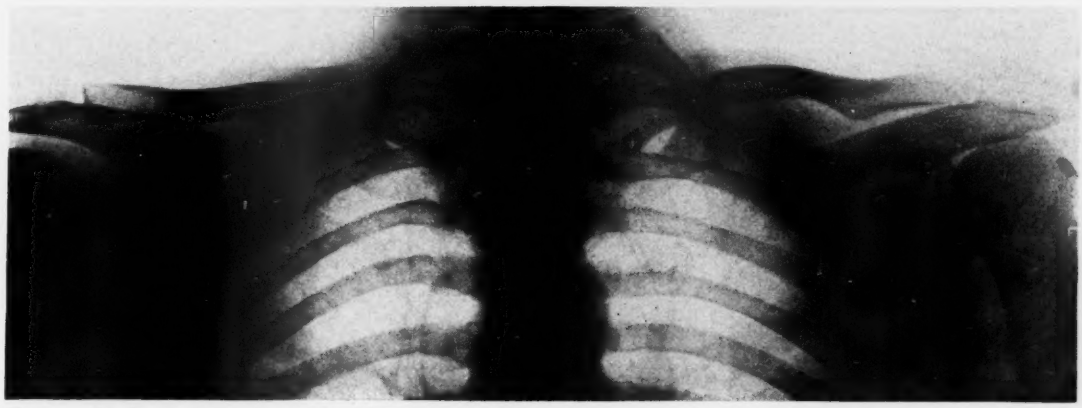

Fig. 12. H. R. K. Visualization of full length of clavicles in spite of size of subject (male, age 32, weight 220 pounds).

SUMMARY

A technique for the study of clavicles, acromioclavicular joints, sternoclavicular joints, and most shoulders is offered, yielding an undisputable control study; in the vertical, most usual functioning, position; yielding comparative length studies of end results of fractured clavicles; with the minimum amount of film expense, and requiring no special apparatus in its employment.

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THE X-RAY IN THE TREATMENT OF INFECTION*

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NOTES ON LITERATURE BY HARRY F. MERSHON, M.D.

AN infection, according to Webster is "the state produced by the entrance into, and multiplication within the body, of pathogenic organisms." Therefore, an inflammatory lesion is "a morbid condition consisting in congestion of blood vessels and exudation of plasma and blood corpuscles, at the site of an infection or injury; manifested outwardly by redness and swelling, attended with heat and pain."

Observations on bactericidal response to solar radiation dates back to 1877. Much of the earlier work dealt with bactericidal or lethal effects. Sunlight was first studied, and the observations of Downes and Blount constitute the first discovery of the killing action of light on bacteria.[1] Notable impetus was given to investigations dealing with radiation, by the Finsen Institute of Copenhagen, established in 1896, using ultraviolet. Considerable study was done by various workers on the effectiveness of various wave-lengths. Bie[2] regarded all wave-lengths as inactivating but shorter wave-lengths as more effective. Other conditions studied regarding the effectiveness of radiation included temperature, hydrogen ion concentration, the medium, and its absorption of radiant energy. Some differences in the sensitivity of various species of bacteria to the lethal effect of fight have been consistently reported.[3] Later spark sources of radiation with various metals as terminals were employed.

The earlier work on the effects of *x*-ray on bacteria was largely negative. Minch, in 1896, was probably first to undertake a determination of its efficiency as a bactericidal agent. Most of the work was done on strains of bacteria which were streaked on the surface of agar plates, then exposed. No effect of radiation could be detected from the gross appearance of colony development.[4]

Later results were obtained which showed that while the irradiation of inoculated animals might increase their resistance to the disease normally induced, there was no evident effect on the morphology or physiology of the bacteria, when irradiation on these organisms was carried out independently.

Unfortunately, most of the quantitative *x*-radiation work on bacteria relates to lethal effects only. From all data now available it is well known that if any population of microorganisms is exposed to radiation inducing lethal effects, one may plot the percentage of survivals against the time of exposure and the results will appear in the form of a logarithmic or S-shaped curve.[5]

From the State Roentgen Radiological and Cancer Institute[6] we learn that serum from fifty patients and an unstated number of rabbits which had been inoculated with staphylococcus, was studied to determine the quantity of antibodies before and after *x*-ray therapy. Four reactions were studied —antistaphylolysin, bacteriolysin, lipoid reaction, and the opsonic reaction of complement fixation. The investigation showed that large quantities of antibodies accumulate in the organism during development of the inflammatory process, and that *x*-ray does not increase their number. Only the opsonic index showed a slight increase. These authors believe therefore that the benefit from *x*-ray lies in hyperemia and intensified lymph circulation (Motohina) which brings large quantities of antibodies from other parts of the organism to the

* From the Oncological Service of Drs. Soiland, Costolow and Meland.

inflammatory focus. *X*-rays have no direct effect on microorganisms, but they produced a distinct effect on mixtures of

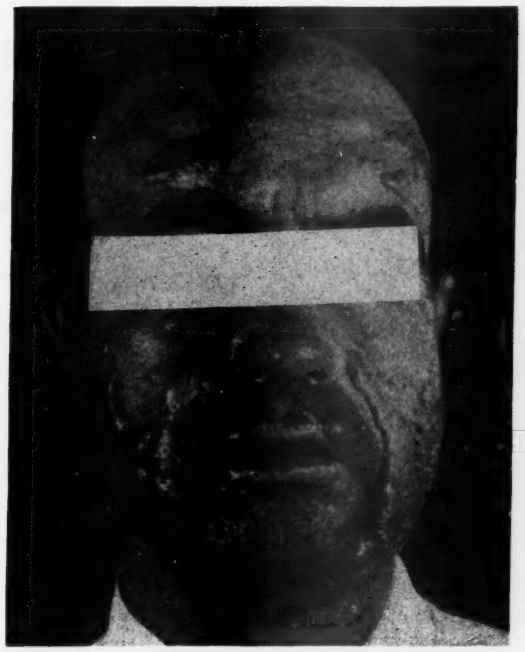


Fig. 1. Case 1. A medical colleague—badly burned from fire on head, face, chest and arms, with subsequent keloidal scars and accompanying infection and inflammation. Before treatment, September 1934.

microbes, serum, and leucocytes, stimulating phagocytosis.

Carl Fried[7] reports on experimental pneumonia in guinea-pigs. Macroscopically, the lungs of the control animals showed complete hepatization, while the lungs of the irradiated animals appeared almost normal, with only scattered areas of hepatization. Microscopically, the irradiated lungs disclosed less congestion, edema, infiltration and abscess formation than the non-treated lungs. An essential finding was the vicarious emphysema, partly lobular, partly marginal, in the irradiated animals.

The use of Roentgen rays as a means of treatment in acute infections is far from having the place in therapy which the results justify. While *x*-rays themselves do not possess the bactericidal power, they do stimulate within tissues the responses of hyperemia along with destruction of leucocytes and lymphocytes, thus liberating endotoxins which possess the power of phagocytosis. Both high and low voltages may be used, dependent upon the nature of the lesion. The dosage is small, repeated at

Fig. 2. Case 1. Showing keloids of arms, September 1934.

intervals of several days to several weeks. Often a marked reaction with increasing rise of temperature is contraindication to further irradiation. The response to treatment is slow and continuous, and the repetition of therapy must not be too rapid, for a toxic condition might be excited.

Kaplan[8] comments on the following conditions treated:

1. Tubercular peritonitis. Ascites is usually present and this is best first drained, and the abdomen then irradiated.

2. Tubercular joints. Reduces the swelling and pain and helps to restore motion.

3. Tubercular glands. These usually occur in the neck, occasionally in the axillary and inguinal areas. They are usually unilateral, but often bilateral. One or many glands may be involved. In the majority of cases with gland involvement the chest is clear. This is reported to be attributed to different strains of the tubercle bacillus organism.

4. Osteomyelitis. Contraindicated in the acute stage. In the persistent chronic con-

ditions therapy helps sequestrum formation with growth of healthy granulation tissue, and after the removal of necrotic

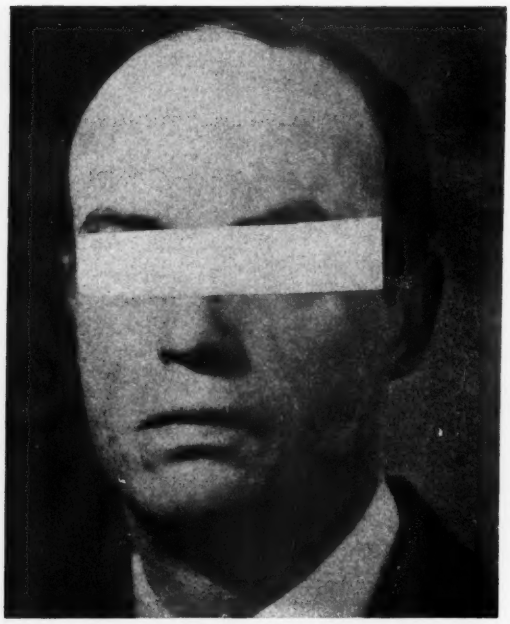


Fig. 3. Case 1, one year later. Treatment was exclusively high voltage *x*-ray.

bone, stimulates the formation of reparative scar. Chronic sinuses are stimulated to healing by the *x*-rays.

5. Gonococcal arthritis. *X*-ray of value in semi-acute stage. Reduces the pain and swelling and allows for motion.

Garland[9] reports favorable results with small doses of *x*-rays in both acute and chronic arthritides of nonspecific nature, as well as of gonorrheal origin. Kelly and Dowell[10] state that in gas bacillus infections, radiation therapy is beneficial. Powell[11] gives an interesting record of *x*-ray therapy in pneumonia of Types I, II and III, with satisfactory results. He calls attention to the rather rapid fall in temperature with a feeling of well being and early return to normalcy. He warns that in post-influenzal pneumonia *x*-ray treatment is contraindicated should leucopenia coëxist. Liljedahl[12] feels that radiation therapy in superficial pathology is now quite safe due to present day knowledge of physics, measurements and dosage. Among the diseases in which he recommends radiation therapy the following predominate: Boils, furuncles, carbuncles, erysipelas, surgical parotitis, and tubercular adenitis. He sums up as follows: "All infective processes do not respond, and as in all forms of treatment some cases are more or less refractory. In these instances radiation should not be persisted in but other measures substituted. Radiation has a beneficial effect in acute infective processes and is worthy of trial and consideration."

Our own experience with inflammatory lesions of the skin—such as acne—dates back to 1901; at that time we probably obtained more damage to the skin than benefit. However, with the passage of time and the accumulation of more knowledge and experience, the situation improved materially. It has been impressed upon radiologists from a great many sources that radiation therapy is contraindicated in the presence of acute infections and inflammatory lesions. Clinical experience and reports from all over the world have emphatically exploded this admonition. In fact, such acute inflammatory conditions as erysipelas, carbuncles, herpes zoster, etc., respond with astonishing rapidity in direct ratio to their pathologic activity. True, many failures are also recorded, but in no greater proportion than are found with many remedies known as "specifics" for these and other recognized maladies. From a clinical angle the subject of radiation therapy in infectious disease covers an enormous expanse of human pathology.

As early as 1902 we were treating localized tuberculous disease, such as lupus vulgaris, tuberculous synovitis, and adenopathies, also joint tuberculosis. The static machine at that time with its relatively high voltage and low amperage seemed especially fitted for this type of work, and while we blundered along without adequate knowledge of dosage or protective factors the results as a whole were encouraging. I remember in 1905, when visiting the Finsen Light Institute in Copenhagen for the first time, being forcibly struck by the excellent work accomplished there with the ultra-

violet, quartz and carbon lamps then in vogue. While there is quite a gap between the ultraviolet and *x*-ray beams on the scale of light energy, both of those agents have their place in dealing with inflammatory and infectious disease.

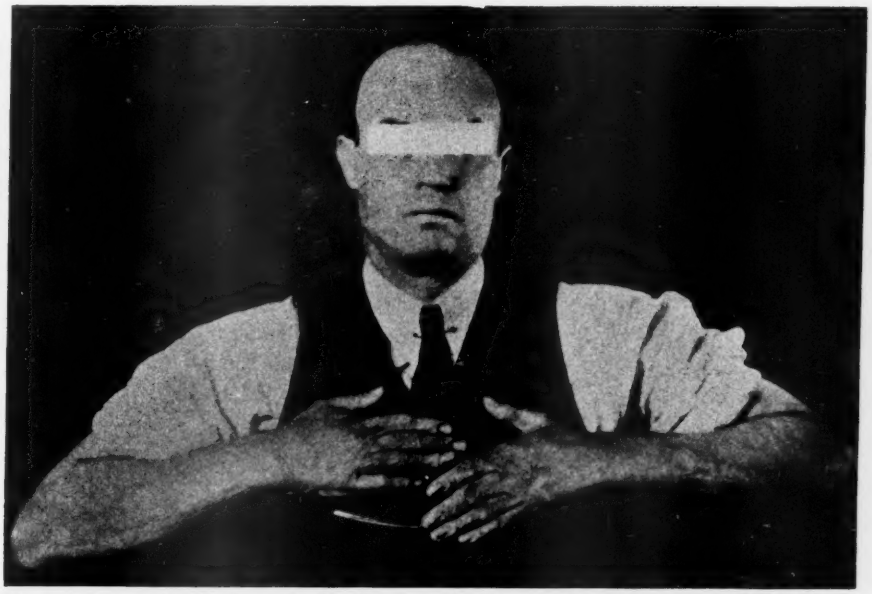


Fig. 4. Case 1, April 26, 1938, showing practically complete disappearance of lesions.

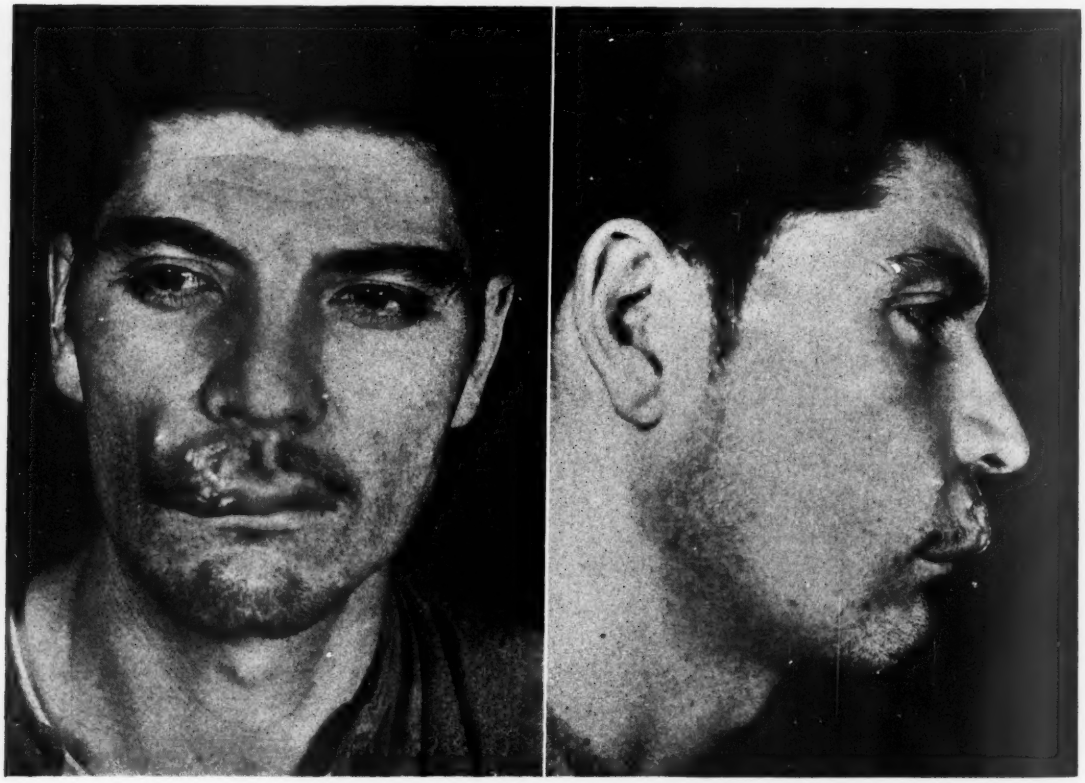
One hears frequent warnings never to use the *x*-ray in the presence of an acute inflammatory lesion. This is utter nonsense, for one can name thousands of instances where, under the careful use of *x*-rays, innumerable patients suffering from the most painful types of inflammatory disease, obtained astonishingly rapid relief. It is necessary only to mention examples such as acute carbuncle, poison ivy, herpes, sycosis barbae, erysipelas, and that despairing condition of malignancy—cancer en cuirasse of the female breast. Here we have a selection of conditions, both specific and non-specific, which overwhelmingly refute the citation alluded to above.

I recall one paradoxical situation concerning a very distinguished dermatologic colleague, whose textbook on skin diseases has a wide distribution. It is a masterful treatise and authoritive from every angle. On the occasion of a visit to this good friend I called his attention to the book, inquiring why he devoted over a thousand pages to classical description of each and every type of dermatologic pathology with elaborate formulae of unguents, lotions, powders of every kind and description for treatment, and only casually mentioned radiation therapy. Then to add a little sauce to the pudding, I pointed out the battery of six *x*-ray therapy machines merrily humming in his office at one and the same time. Strange as it may appear, we are still the best of friends while not infrequently crossing swords over this delicate subject.

In looking over past case records in our own service, the following formidable array of pathologic conditions has been submitted to radiation therapy: osteomyelitis —chrondritis; dermatophytosis; acne vulgaris; furunculosis; carbuncles; ringworm; sycosis barbae; paronychia; lupus vulgaris; lupus erythematosis; erysipelas; mycosis fungoides; molluscum contagiosa; granuloma inguinale; trachoma; seborrheic dermatitis; otorrhea; mastoiditis; rhinophyma; acne rosacea; rhinoscleroma; verrucae; actinomycosis; coccidiosis; inflammatory tonsils; pertussis; pneumonia; empyemic sinuses; bronchiectasis; mastitis; pruritus; condyloma; chronic salpingitis; tubercular salpingitis; encephalitis; brachial neuritis, sciatica and radiculitis.

A B

FIG. 5. Case II. A, on October 11, 1937. Ulcer overlying tibia, following scraping of leg against a barnacle-covered wharf. B, on June 11, 1938, following *x*-ray therapy.



A B

FIG. 6. Case III. A, on June 10, 1938. Furuncle and cellulitis of right upper lip following sqeezing of a pimple. B, same in profile view.

At the Los Angeles General Hospital the newly installed radiation therapy department is now functioning under the supervision of Dr. Ray Carter, the Head of the Department, and our own clinical associates, Dr. William E. Costolow and Dr. Harry F. Mershon.

A number of dermatologists, and not a few radiologists, advocate unfiltered *x*-ray therapy. While in skilful hands and under strict regulation protective factors, this is permissible, yet in the writer's opinion the use of raw radiation should be discouraged. There are altogether too many chances for excessive dosage from the unscreened tube, which can so easily be avoided by employing selective filters. In fact, the tendency of today is to step up both voltage and filter factors, not only for depth dosage but in skin and superficial disease also. This has been strikingly demonstrated in our last few years' work upon extensive surface lesions wherein supervoltage therapy was successful in clearing up certain conditions which had become static to cautery and ordinary *x*-ray and radium applications. A few examples are illustrated in the text.

CONCLUSIONS

Inflammatory lesions during the past decade have been subjected to intensive study in their relation to radiation therapy. The knowledge accumulated by correlating the experience and research work of biologists, physicists, radiologists and clinicians is rapidly standardizing this comprehensive branch of the medical sciences. It is therefore quite possible to anticipate the early arrival of even more precise methods of treatment which will eliminate most of the confusion, ignorance and hazards which have surrounded radiation therapy since its inception.

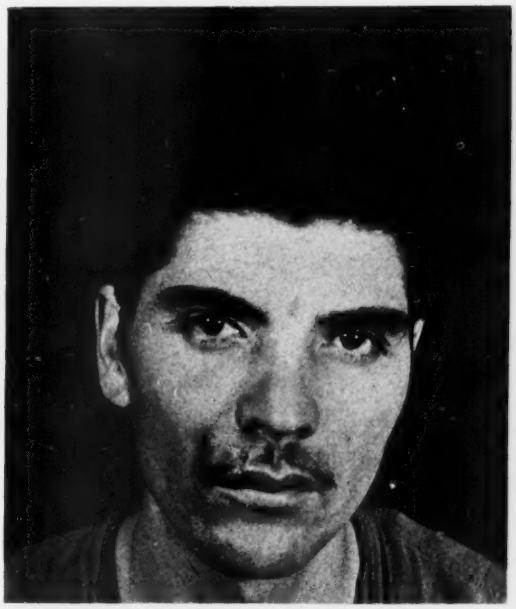


Fig. 7. Case III, on June 17, 1938, following three treatments of low voltage *x*-ray, 75 R each, 4 mm. aluminum filter.

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AGGRAVATION OF CHRONIC PULMONARY TUBERCULOSIS BY TRAUMA OF THE CHEST

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ANY deleterious effect of trauma of the chest on underlying pulmonary tuberculosis is usually shown by the activation of one or more of the mechanisms which operate in the natural course of the disease. In the majority of cases the inclination of the patient to link some symptom or train of symptoms to a specific event is found by the medical study not to be justified. This is true whether the suspected activating cause be trauma or some other extraordinary happening. Nevertheless, there are situations in which the trauma can reasonably be implicated. Many times the decision is rather a delicate one and on this account there is a heavy obligation on the physician to be completely objective in his observation and to base judgment on thorough knowledge and experience. The following is a discussion of some of the fundamental principles which have been found useful as the basis of thinking and action in this field.

OUTLINE OF PATHOGENESIS

It is not necessary to discuss the earliest origins of the tuberculous lesion in the human body since we are here concerned with established pulmonary disease. It is sufficient to point out that, in infants and young children, clinical tuberculosis tends to spread through the body by the lymph and blood streams and, when fatal, to be represented by tuberculous meningitis or acute generalized miliary tuberculosis. It is in adolescence and early adult life that the pulmonary form of the disease begins to show itself, and a study of vital statistics in any community will demonstrate that sickness and death from the disease during and after these age periods is with relatively few exceptions traceable to pulmonary lesions and their complications.

The commonest early lesion in clinical pulmonary tuberculosis is a small infiltration (pathologically a patch of tuberculous bronchopneumonia) located usually in the upper part of one lung, often below the apex. At this stage physical signs as a rule are lacking, symptoms are absent or very vague, tubercle bacilli are not discharged in the sputum, but the roentgenogram shows a small patch of mottling. Healing when it occurs is by resolution and fibrosis, usually leaving behind a small nodular or stellate scar. The whole process up to this point may be unknown to the patient. Unless treatment is given early, however, the predominant tendency of the early infiltration is to spread. This consists of peripheral extension of the inflammation in many cases and, most important, central caseation and liquefaction. With the establishment of central necrosis, ulceration into the bronchial tubes eventually follows; this event frequently marks, on one hand, the first recognition of symptoms and, on the other, the initiation of chronic disease. The process is no longer confined to the immediate environs of the first infiltration because the escape of infected discharges permits their inhalation into more distant parts of the same or of the opposite lung, explaining the so-called bronchogenic dissemination phase in the pathogenesis. New lesions, thus established, may progress just as the early infiltration did, eventually leading to multiple and coalescing cavities in one or both lungs. The speed and intensity of these processes vary greatly. They may be slow and insidious or rapid and clinically acute, accounting both for

the chronic case drawn out over many years, and for death within a few months after the appearance of the first infiltration. This depends somewhat on constitutional factors, acuteness being observed more often in young people, especially young girls, in old debilitated people, people suffering from uncontrolled diabetes, and in negroes. Accidental intrinsic factors play an important role. Thus, a hemorrhage from a small fresh cavity may lead to acute and fatal dissemination of the infection. Interruption or arrest of the progressive processes may occur at any phase, and this, if permanent, leaves behind fibrosis which, if extensive, produces more or less disabling mechanical effects.

In contrast to the behavior of the disease in young children, where the caseating lesions most often are found in the mediastinal lymph nodes leading to lymphatic and hematogenous dissemination, in the chronic pulmonary case, the regional lymph nodes are not heavily involved and complications are related most often to a contamination of the surfaces of respiratory and alimentary canal by the infectious discharges from the pulmonary cavities. Accordingly, frequent complications are tuberculosis of the larynx and tuberculosis of the intestines. Pleurisy, usually of the fibrinous, plastic and adhesive type, is found over the involved part of the lung; and proximity of a lesion to the pleura sometimes leads to an ulcerative perforation of this structure, resulting in pneumothorax with empyema. It must be borne in mind, however, that a well healed fibrous tuberculous lesion is usually associated with surrounding alveolar emphysematous changes. If these are superficial and bullous in nature, the pleura becomes thinned out and sometimes gives way, resulting in spontaneous pneumothorax. This differs in significance from that caused by the other mechanism in that empyema usually does not follow and healing of the tear with reëxpansion of the lung usually occurs without any lasting disability. Hematogenous lesions may also be seen associated with chronic pulmonary tuberculosis (e.g., genitourinary tuberculosis, tuberculosis of the bones, etc.).

SYMPTOMS IN RELATION TO PULMONARY LESIONS

Clinical judgment depends greatly on the proper correlation of symptoms with the state or behavior of the pulmonary lesions. Except in the chronic arrested case with secondary bronchitis, cough is usually not a feature unless there is an ulcerating pulmonary lesion emptying its discharges into the bronchi. Resulting irritation produces a cough which should be productive of sputum. Sometimes, however, the patient swallows the discharges which rise in his throat and reports no sputum. This explains why tubercle bacilli in such cases are not recovered except by examination of the gastric contents. The sputum, when it comes from a freshly ulcerating lesion, is greenish and mucopurulent. As the process lessens the sputum becomes yellowish, more mucoid, and finally consists only of mucus with small flecks of yellow pus. The quantity of the sputum varies in relationship to the activity of the necrotic process.

Determination of the presence of tubercle bacilli in the sputum is extremely important. First it is necessary to make sure that the patient submits a specimen actually raised from the trachea; second, it must be appreciated that the numbers of bacilli present in specimens submitted vary greatly. If numerous, they are detected by examination of the routine direct smear. If scarce, they may be found only by special concentration, culture or animal inoculation methods. Consequently the laboratory report should always state the method of examination of the specimen. In practically every case with pulmonary cavity the sputum is found to be positive.

Hemoptysis. With few exceptions, bleeding originates from the erosion of blood vessels in a cavity communicating with bronchi, in which case the sputum will be found positive for tubercle bacilli. Occasionally bleeding occurs in rather extensive fibroid cases without cavities and

is traceable to the rupture of superficial vessels in the bronchi. This mechanism cannot be assumed until careful examination shows that the sputum is negative for bacilli.

Fever. Persistent fever means active inflammation and absorption of toxins into the blood stream and usually is observed in association with progression of the pulmonary lesions. There may be progressive excavation, however, and sometimes a slow extension of the infiltration without fever. Persistent fever in the presence of a definitely stable pulmonary lesion suggests some complication of a tuberculous nature elsewhere in the body and this must be sought for. There is always a reason for fever.

Night sweats seldom occur without fever and usually are not reported in the early case. Like a rapid pulse they are related to toxemia.

Pain. There is no more misleading symptom than pain. When acute and aggravated by breathing, it is usually explained by associated pleurisy. In old standing cases, even in those which are well arrested, it is the rule rather than the exception for patients, particularly when fatigued or chilled, to experience at times sore aching pains in the chest. These cannot be interpreted as symptoms of activity and the patient as a rule can be reassured.

In order to give symptoms their proper value, especially when the question of trauma is involved, it is necessary always to attempt to identify the underlying mechanism.

POSSIBLE EFFECTS OF TRAUMA OF THE CHEST

The protection of the thoracic contents by the chest wall is effective in the usual circumstances of life. Even when the lungs become tuberculous it is exceptional that this fails. If it does, the effects of trauma may be direct or indirect. Trauma having direct effect is most often of the nature of a blow on the chest inflicted by some moving object or sustained in a fall. The resulting injury may be a simple contusion with or without fracture of the ribs or other bones, or may be more serious due to crushing pressures, etc. Strain from the lifting or carrying of heavy weights is sometimes involved. Penetrating wounds of the thorax with injury of the pleura and lung may be observed. Other alleged causes include explosions and the inhalation of poisonous gases.

In considering the possible effects, the status of the preëxisting tuberculosis should be known, if this is possible. Well arrested fibroid tuberculosis usually tolerates the effects of trauma remarkably well. In other types of cases the intactness of the healing is so incomplete that it can be rather easily interrupted or nullified. In still other cases pulmonary lesions are active, perhaps without the patient's knowledge, or so close to the verge of activity that slight aggravation may easily intensify or accelerate the processes. Each case therefore must be judged strictly according to its own circumstances. The following principles are of value for this purpose:

A direct blow on the chest or a severe strain may precipitate the rupture of a vessel in a tuberculous cavity. The flowing blood may wash tubercle bacilli from the cavity wall and then may be inhaled into other parts of the lung, when the resulting infection often sets up new secondary lesions. Similarly, a blow or a strain may precipitate the rupture of the pleura already weakened by a subpleural caseous process or by emphysema as described above. The patient then may immediately experience alarming symptoms of pneumothorax, or there may be only a small pleural leak leading to gradual collapse of the lung and symptoms of slowly increasing severity. These happenings have a close time relationship with the trauma and are apparent, as a rule, immediately or within a few hours. Very occasionally, injury of the chest wall, particularly if the ribs are fractured, sets up active tuberculous pleurisy which may be associated with serous effusion and run the usual febrile course.

More rarely still, the injury may cause leakage of blood into the pleura, and the resulting hemopneumothorax may become secondarily infected by tubercle bacilli. In such cases the prolonged febrile course suggests that the symptoms are not related merely to simple hemothorax. In any type of pleurisy the problem is to distinguish the simple traumatic inflammations from those which are complicated by the presence of actual tuberculous infection.

It is more difficult to trace an acceleration or initiation of pulmonary inflammation to direct blows or strains when events such as those described above do not declare themselves promptly. It is conceivable that a severe blow or a crushing injury may have a mechanical effect on a tuberculous lesion aggravating the inflammation, but I have never been able to identify this as a factor in an early tuberculous infiltration and seldom in a well arrested fibrous lesion. In smoldering cases with extensive lesions and cavities, occasionally the prompt development of increased fever and increased cough and expectoration has made justifiable the assumption that there is a definite relationship. Usually the effect has been only temporary, later events being attributable to the natural course of the disease.

A special word may be said about puncture wounds involving an injury of the pleura and lung, such as bullet wounds or perforation by a fractured rib. The extensive experience of the World War and that obtained from other sources indicate that injury of the healthy parts of the lung is not likely to aggravate tuberculosis in other parts. Penetration of the tuberculous part, however, may produce aggravation. Likewise, creation of a pleural perforation with pneumothorax or hemopneumothorax may be deleterious if tubercle bacilli escape into the pleura from the lung, leading to empyema.

The experience of the World War also proved groundless the fear that the inhalation of poisonous gases might often lead to the aggravation and intensification of tuberculous lesions. The most tangible effect of such inhalation is a temporary catarrhal inflammation of the respiratory mucous membranes and, in severe cases, more profound nonspecific inflammation leading eventually to fibrosis and emphysema. Conceivably, severe catarrhal inflammation from gassing might cause a bronchogenic spread of tuberculous infection from a preëxisting cavity, but I have never identified such a happening.

Among the indirect effects which trauma may have is that related to intercurrent nontuberculous infection. Occasionally such a nonspecific inflammation may have a relationship with trauma and in turn lead to an activation or aggravation of the tuberculous lesions. Certain general observations should be noted here. In the first place it is remarkable how well many tuberculous patients tolerate pneumococcic lobar pneumonia. It is seldom that an arrested case is aggravated by the pneumonia. The hazard is somewhat greater in bronchopneumonia due to streptococcus, staphylococcus or anaerobic organisms which tends to go on to abscess formation. If the necrotic process in the pulmonary parenchyma extends so as to take in a section bearing tuberculous lesions, tubercle bacilli are likely to be liberated and may be carried into the healthy parts of the lung in the purulent discharges which sometimes are profuse. Such instances necessitate proof that the bronchopneumonia was related to the trauma. This is possible, for instance, if during an accident which may or may not involve the chest, the person aspirates a foreign body or a tooth which may have become dislodged. Severe blows on the chest, particularly if the ribs are fractured, may in exceptional instances cause a congestion and suffusion of serous fluid in the lung and this may prove a good medium for secondary infection with organisms from the mouth or respiratory tract, resulting finally in suppuration. Such a succession of events is favored when a period of unconsciousness follows the trauma, since this permits material more

easily to be aspirated into and retained in the bronchi long enough to set up inflammation. Such a mechanism may also be involved in submersion.

Inflammation of the pleura following a severe blow on the thorax, especially if the ribs are fractured, is not uncommon. Usually it is of short duration, produces no fever or only a transient fever, and heals rapidly, perhaps leaving behind demonstrable adhesions. This alone cannot be interpreted as evidence of aggravation of the tuberculosis; and the pain which sometimes persists for a good while after the trauma should be interpreted very carefully in view of the probability that it is in no way related to the tuberculosis.

A rare effect of trauma of the chest is evidenced by subsequent dissemination of infection through the blood stream, leading to isolated local lesions such as tuberculosis in a joint or more generalized manifestations. As mentioned, hematogenous disseminations of serious consequence are uncommon in chronic pulmonary tuberculosis, and it is seldom that such a mechanism, when present, is related to trauma.

Rather often the question comes up whether the lowering of the general resistance from the damage caused by trauma is responsible for the subsequent aggravation of pulmonary tuberculosis. Undoubtedly such effects are conceivable, and one must view this as he would a debilitating nontuberculous disease which sometimes opens the way for tuberculosis. However, such an assumption is seldom to be considered unless there is a reasonably close time relationship between the trauma and the aggravation of the tuberculosis. As a rule this should become evident under proper clinical observation within a month. The time limits are arbitrary, and judgment should be supported by other observations such as an unusual change in the behavior of the lesions following the trauma. In practice the limits of reason are frequently exceeded in trying to establish such a relationship where compensation is involved.

One cannot ignore the psychic factor in patients who have suffered trauma. For the first time the patient may become aware that he has tuberculosis and for the first time this may concentrate his attention on manifestations which were present before but not recognized. In medicolegal cases the uncertainty of the outcome of the claim, the variance of the opinions of different physicians, the necessity of losing employment and the mystification of encountering the disease for the first time may produce many psychic disturbances and eventually help to undermine the resistance against the disease. Unfortunately our practices in compensation court often ignore this factor and sometimes, therefore, accomplish more harm than good.

JUDGMENT OF THE EFFECTS OF TRAUMA IN THE INDIVIDUAL CASE

Innumerable possibilities of various mechanisms of pulmonary tuberculosis make it unwise to try to formulate general and inflexible rules of judgment. It is the obligation of the physician, therefore, to collect carefully all the necessary data so that he may form his judgment with the maximum of objective evidence. The necessary thorough study of each case usually is best conducted in a hospital or sanatorium. Attention should be given particularly to a complete and accurate history of symptoms before and after the trauma. Complete physical examination should include roentgenographs of the chest in every case of trauma involving the chest, whether or not tuberculosis is suspected. If the patient has had roentgenograms previously, these should be procured for comparison. The sputum should be collected in containers so as to show its gross characteristics and should be submitted to repeated and special examination for the presence of tubercle bacilli. Studies of the blood cells and of the sedimentation rate of the erythrocytes should be made with the understanding that these may have no

bearing on the question but in some cases may be of considerable importance. The complete examination should include a search for extrapulmonary tuberculous lesions, especially lesions of the larynx, intestines and pleura. Pleural effusions should always be tapped and studied, especially for the detection of tubercle bacilli, usually with animal inoculation. In most cases a period of observation of weeks, or perhaps several months, will be required to determine fully the effects of the trauma and the necessity for further treatment.

After determining the nature and the extent of the lesions, the problem to decide is whether their present behavior can be explained logically according to the usual pathogenesis of the disease or whether it has been modified in any way by the trauma. Clear cut judgment requires that the evidence be definite and demonstrable and the time element should be given great weight. Reliable opinions can hardly be expected unless the physician has a background of experience with the disease. If he has not, he should ask the consultation of an expert.

DURATION OF EFFECTS OF TRAUMA

This again is a matter of judgment, the reasonableness of which depends more on objective findings than on subjective complaints. Traumatic pneumothorax due to rupture of a subpleural emphysematous bleb usually does not cause long standing disability, since reëxpansion of the lung leaves conditions as they were previously. On the contrary, the same condition caused by ulcerative perforation of a caseous lesion through the pleura is usually followed by empyema or effusion and often causes prolonged or permanent disability. Similarly, hemoptysis from the simple rupture of a superficial vessel in a bronchus may be only a temporary and passing event causing no real aggravation of the tuberculosis, while bleeding from an open cavity may promptly produce bronchogenic extensions of the disease with serious consequences. The importance therefore of identifying and evaluating the underlying mechanisms is clear.

TREATMENT

Aggravation caused by trauma calls for treatment, the same as when such a happening is related to other causes. Rest in bed is indicated for all cases in which the symptoms suggest activity of the disease, whether or not an effect on the pulmonary lesions is immediately demonstrable. Subsequent observation, particularly with serial roentgenograms of the chest, usually shows clearly whether treatment should be continued. It must be emphasized that symptoms alone are not sufficient to decide this point. In cases with hemoptysis, the site of bleeding is to be identified and, if rest and medication do not suffice, artificial pneumothorax must be considered. In cases of traumatic pneumothorax the patient's life may be endangered immediately or later by the accumulation in the pleural cavity of air under pressure. Aspiration of the air and proper treatment in a hospital or sanatorium are of vital importance. When the lung tissue has been damaged by penetrating wounds and blood accumulates in the pleura, the usual treatment is to aspirate blood and replace the air, allowing the lung to remain collapsed until the tear is healed. Transfusions, sometimes of the patient's own blood recovered from his pleura, are often necessary. Tuberculous pleurisy is an active lesion and usually requires bed rest for several months and sanatorium care for another three to six months during which time thoracentesis for withdrawal of the fluid may be indicated.

DURATION OF SANATORIUM CARE OR ITS EQUIVALENT

The proper time to allow return to work and the kind of work which patients may be permitted to do are matters for judgment based on experience. As a rule it may be said that most patients should not resume their usual work until the evidence points to thorough and durable arrest of

their lesions. The tendency is to give longer periods of treatment, that is, from one to two years, in order to avoid disabling relapses and early fatalities. Once the arrest is obtained, the patient's subsequent fate depends largely on his intelligence, his social and economic status, and his mode of life. Employment is only one phase of his living and it is just as important for him to give similar attention to his activities outside his working duties.

SUMMARY

An understanding of the possible effects of trauma of the chest on underlying pulmonary tuberculosis depends primarily on a comprehension of the pathogenesis and of the mechanisms operating to cause symptoms. These are described briefly and it is explained how the pathogenetic factors are sometimes set in motion or aggravated by trauma. The necessity of a thorough study of the individual case and of procuring all possible objective evidence is stressed. Experience with the disease is held to be necessary for the proper judgment of the effects of trauma and of the duration of these effects, drawing fine distinctions according to the nature of the underlying mechanisms. The principles of treatment are mentioned briefly and in general are the same as those applying to clinical tuberculosis whether or not trauma is involved.

DÉBRIDEMENT [in compound fractures] prevents the development of gas bacillus infection and gas bacillus serum is not needed as a prophylactic agent.

GAS GANGRENE

AN ANALYSIS OF 109 CASES ENCOUNTERED IN CIVIL PRACTICE*

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I. INTRODUCTION

GAS gangrene (Humphreys) is an anaerobic, regional, necrotizing wound infection which involves muscle especially, though not exclusively. It is characterized by massive edema and gaseous infiltration, with usually a putrefactive odor.

The purpose of this discussion is to reacquaint the profession with this dread disease which occurs far more frequently than is commonly supposed, particularly in city hospitals which carry on an active emergency service. Here are treated serious accidents which occur under conditions comparable to war injuries, such as gunshot wounds and compound fractures, associated or unassociated with amputation. City hospitals contain many patients with gangrenous extremities. Surgery for this condition frequently is complicated by gas gangrene.

The extremely high death rate in this series leads to the inference that as yet the surgical lessons learned during the World War are not sufficiently appreciated. It certainly is true that the recognition of the disease has not been made early enough, and that the treatment has been unsatisfactory. The profession has not kept abreast of the available practical bacteriologic data on the subject.

Surgeons have accumulated a mass of clinical data, but the bacteriologic work on most of their cases has been unsatisfactory because of laboratory limitations. In the course of the last few decades, particularly under the stimulus of World War conditions, bacteriologists have become interested in this subject and have presented their material with a full appreciation of the clinical viewpoints involved. Because we believe that the bacteriologic side of gas gangrene has not been considered sufficiently, we will deal carefully with the bacteriologic principles which underly this condition.

The recent bacteriologic textbooks of Gay (Humphreys' article) and Topley-Wilson have presented excellent reviews of the mechanism of gas gangrene which are the best information available in English. We have drawn freely from these sources, stressing the factors important from the surgical point of view. Gas gangrene lies in a borderline field between bacteriology and surgery. Closer coöperation between these two departments promises progress.

II. HISTORY OF GAS GANGRENE

What we recognize as gas gangrene has been known as a clinical entity since Hippocrates. Ignorance as to its etiology led observers to name the disease after the outstanding clinical features of the cases seen. Maisonneuve in 1853 called it "fulminating gangrene." The following

* These cases occurred in the San Francisco Hospital (Division of San Francisco Department of Health) during the last eighteen years.

year Salleron collected into a series the cases which occurred in the Crimean War. In 1864, Pirogoff named the condition "acute gangrenous edema." Bottini (1871), however, was the first to demonstrate its infectious nature and transmissibility. In 1877, Pasteur and Joubert discovered their anaerobe, "vibrion septique."

The studies of Koch and Gaffky in Germany (1881) resulted in the description of an anaerobe, known as "bacillus of malignant edema," an organism identical to Pasteur's vibrion septique. The work of these men and that of Chauveau and Arloing in France, led to a conception of a specific disease, caused by a specific microbe. The disease in Germany became known as "malignant edema," and in France as "gangrenous septicemia." The term, "gas gangrene," apparently, was introduced in 1882.

In 1892, Welch and Nuttall discovered another anaerobe, the B. aerogenes capsulatus. The following year, Fraenkel independently described the same organism in several cases of what he called emphysematous phlegmon. He accordingly named it "B. phlegmonis emphysematosae." In 1894, Novy described another anaerobe, "B. novyi." At that time the proper differentiation of these new organisms was so neglected that Veillon and Zuber in 1898 announced the discovery of "B. perfringens," which is identical with the organism described by Welch and Fraenkel. More recently (1915), Weinberg and Séguin described a new organism, the "B. oedematiens," which was identical with Novy's bacillus, described twenty-one years before.

By 1900 Welch's organism had been identified many times in America as the cause of human gas gangrene. Indeed, it was found so frequently in this condition that the organism became known as the "gas bacillus," almost to the exclusion of other anaerobes. In 1913, Cramp in America published a monograph on the subject.

It was, however, the enormous number of fatal cases of gas gangrene during the first years of the World War which led to the most extensive and intensive studies of this disease. In consequence of these researches, the incidence of gas gangrene was checked notably. In 1914, its occurrence among the wounded of the British Expeditionary Force was 12 per cent and the mortality 20 to 25 per cent. By 1918 the incidence had dropped to 1 per cent. In the course of the bacteriologic studies carried on by war-time and later observers, new species of gas-forming anaerobes were discovered. New biologic reactions and new techniques for their study made possible our modern conception of the disease. During these studies, it became apparent that the bacteriologic methods are difficult to master. It was found that many anaerobes—not a specific one—are involved in the pathologic picture. Association of anaerobes with aerobes complicates the problem. Also it became evident that the older bacteriologic studies were not accurate enough for correct differentiation. The outstanding workers in the field of bacteriology of the anaerobic organisms associated with gas gangrene are: Weinberg in France, Zeissler in Germany, Robertson, Henry and McIntosh in England, and Bull and Pritchett in the United States.

III. GENERAL BACTERIOLOGY

Although cases are reported in which aerobic organisms—certain members of the streptococcus and colon groups—produce gas in tissues, practically all gas gangrene is caused by anaerobic bacilli. Usually, however, the anaerobes are in association with aerobes. When anaerobes alone are found, several species generally occur in combination. Weinberg and Séguin (1918) in an analysis of ninety-one cases found anaerobes alone in twenty-four instances. In this group of pure anaerobic infections, there were fourteen cases where more than one species of anaerobes were demonstrated. As many as thirty-eight combinations of anaerobic bacilli occurred in these ninety-one cases. When aerobic organisms are found in combination with

anaerobes, the aerobes complicate the pathologic picture by adding their own destructive toxins. These produce conditions favorable to the multiplication and pathogenic action of the anaerobic organisms present.

The anaerobic organisms found in gas gangrene have been placed in a new genus, *clostridium* (derived from the Greek word for spindle). They are large, thick, Gram-positive, spore-forming rods, characterized by the anaerobic or micro-aerophilic nature

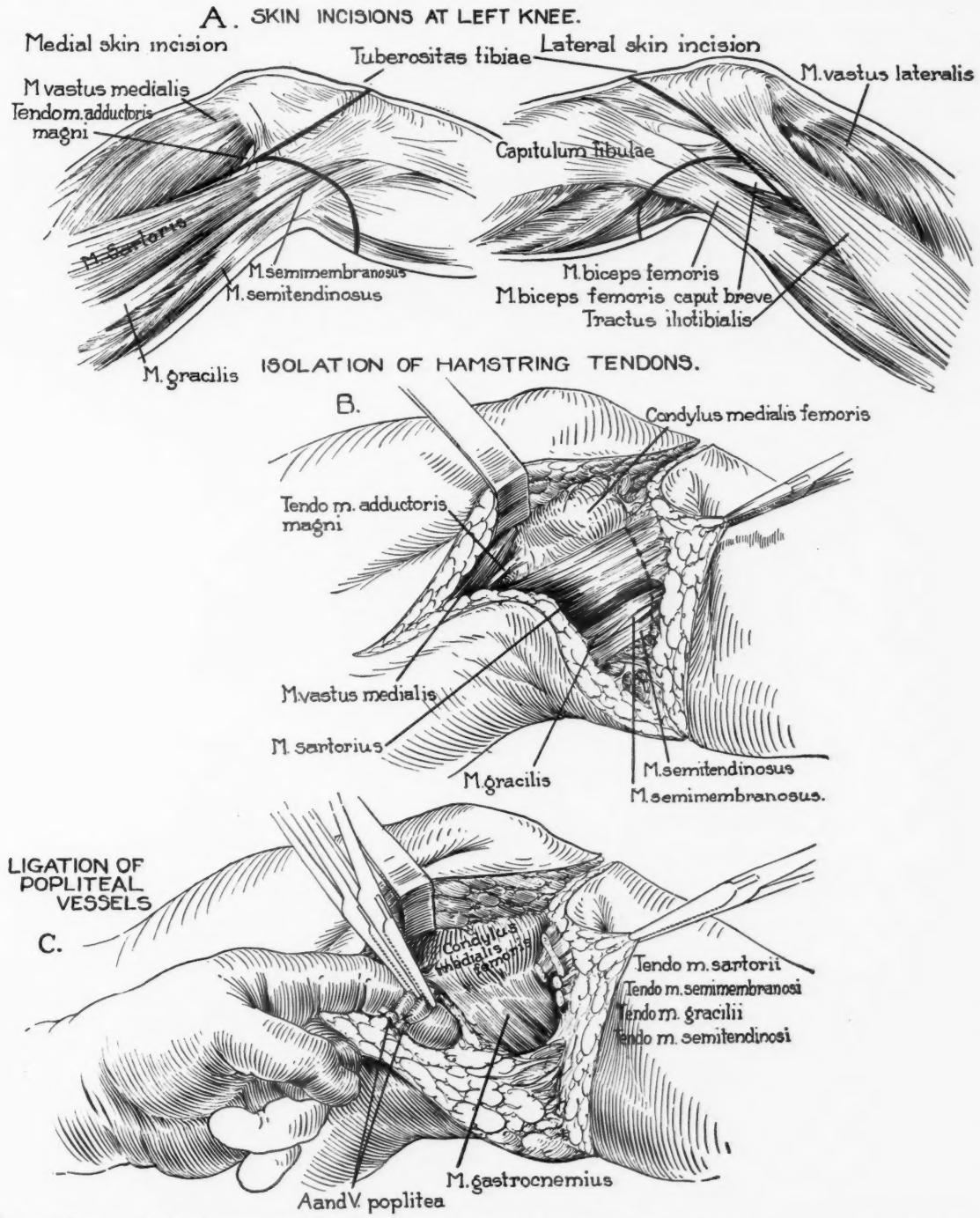


Fig. 1. New tendoplastic amputation through the femur at the knee. A, B, C, stages in operation leading to ligation of popliteal vessels after section of medial hamstring tendons at their insertion on the tibia. (From Callander's "Surgical Anatomy," Saunders.)

of their growth. This means that they are affected so deleteriously by the concentration of the oxygen present in the atmosphere that they are unable to grow or reproduce unless they are protected in some measure from it. They often decompose protein media by enzyme action and usually ferment carbohydrates. Of the 100 or more species belonging to the group of clostridia, very few are toxic and pathogenic for man. In this paper, we do not deal with the neurotoxic clostridia of tetanus and botulism, nor are we concerned with the nontoxic clostridia which

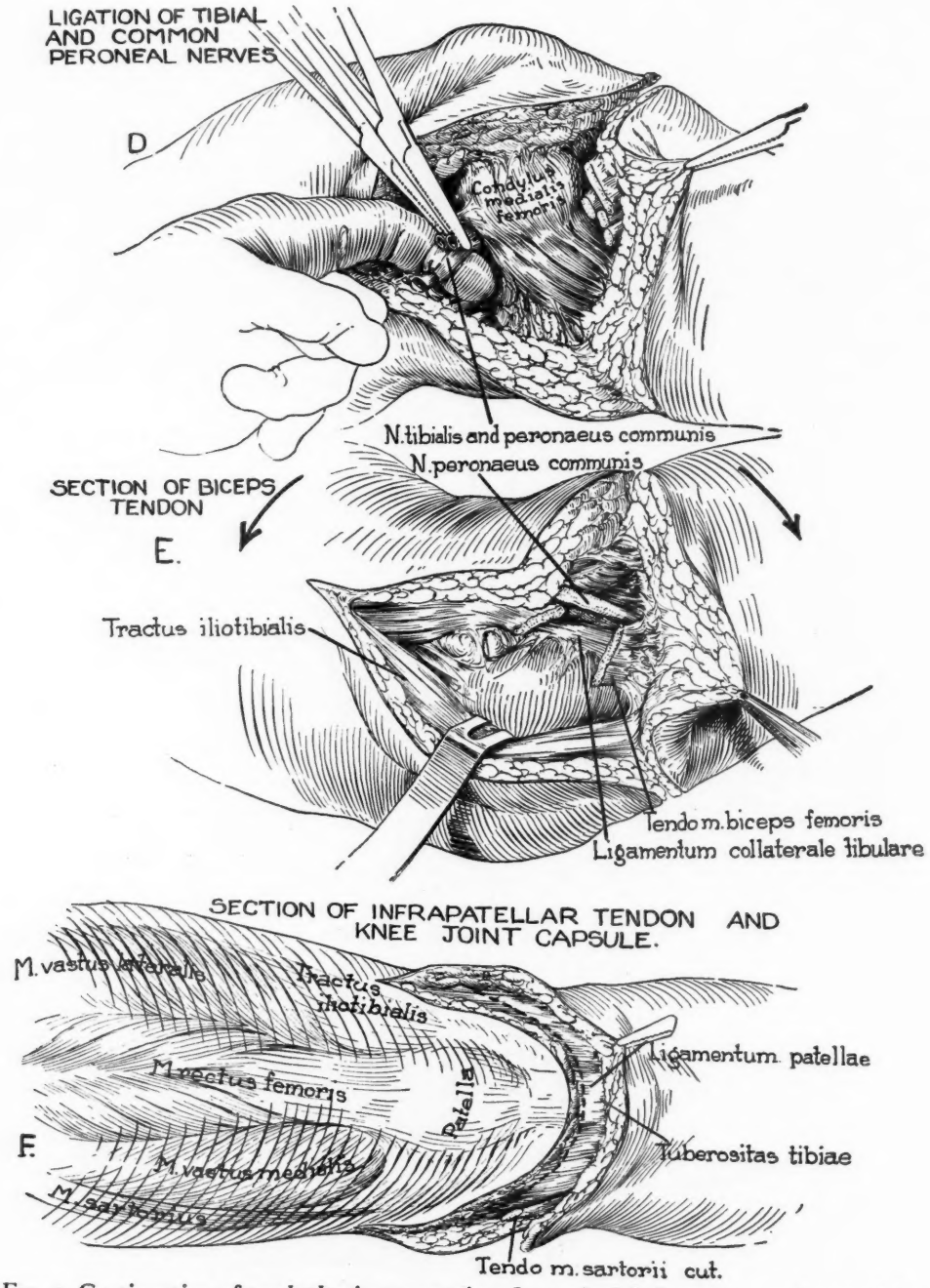


FIG. 2. Continuation of tendoplastic amputation. In E, the left knee is turned toward the operator and the biceps tendon is sectioned at its insertion on the fibula. (From Callander's "Surgical Anatomy," Saunders.)

are common commensals with the histotoxic or edematogenic gas gangrene organisms.

sordelli; and Cl. histolyticum. The characteristics of each member of the histotoxic group will not be discussed here because

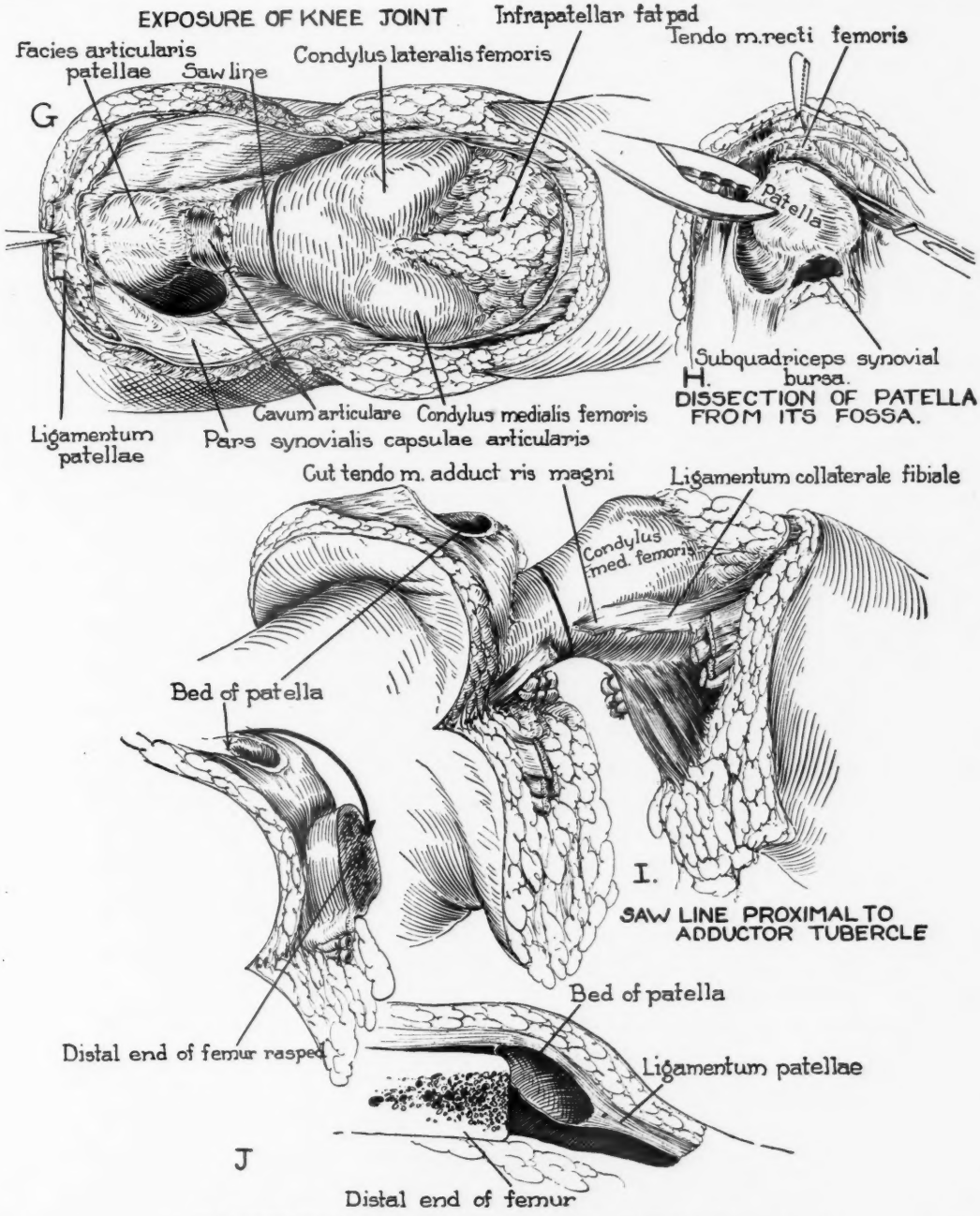
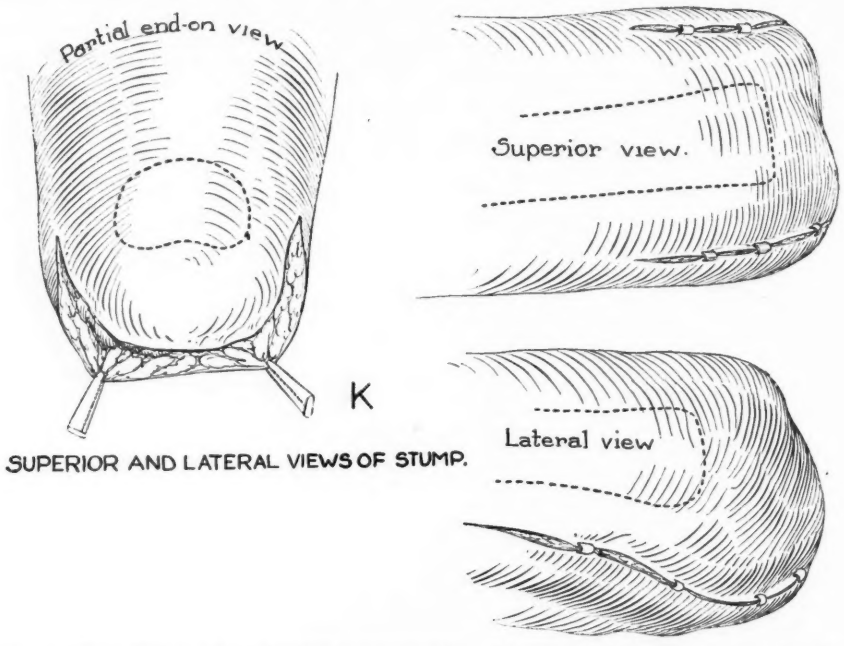


Fig. 3. Continuation of tendoplastic amputation. The patella is dissected from its fossa and the femur is sectioned a varying distance proximal to the adductor tubercle. Note length of amputation flaps. (From Callander's "Surgical Anatomy," Saunders.)

The following clostridia are those commonly cultured in gas gangrene cases: Cl. Welchii (B. aerogenes capsulatus; B. perfringens); Cl. oedematis maligni (vibrion septique); Cl. novyi (oedematiens); Cl. the available bacteriologic methods and facilities as a rule will not permit a sufficiently early identification of anaerobes in gas gangrene cases to be of any value in the clinical management of the infection.

Mention will be made of the important factors which are common to most of them.

In common with the non-pathogenic anaerobes, the histotoxic anaerobes inhabit the soil and infest the lower alimentary tract in man and animals. While their presence in the soil may be the result of fecal contamination, they have been found in virgin soil far from human or animal habitation. It is probable that their primary habitat is the soil. Doubtless they are ingested with vegetables and fruit. Many varieties are able to adapt themselves temporarily or permanently to existence in the intestinal canal.



SUPERIOR AND LATERAL VIEWS OF STUMP.

FIG. 4. Completion of tendoplastic amputation. Note redundant flaps, closed only by few skin clips or sutures. Widely spaced stitches leave ample space for drainage. (From Callander's "Surgical Anatomy," Saunders.)

Concerning the habitat of Cl. welchii, Humphreys has the following to say: "This organism is ubiquitous. Zeissler and Rassfeld found it in 100 per cent of 200 soil specimens examined by them. It has been found with constancy, in varying numbers, in the feces of practically all higher animals. Hence, its spores or seeds are likely to persist indefinitely on anything that can be contaminated by dust or dirt—and this means practically everything. They are common in dairy products and foods of all description; on the skin, clothing, and hair, in the mouth, and even within the parenchyma of the liver and other viscera of normal men, dogs, sheep, and animals in general. Many of these strains are non-pathogenic. On the other hand, many are found, when growing under suitable conditions, to be exceedingly virulent. Bull obtained his most toxic strain from the lining of an old overcoat. We may consider, then, that we have potential incitants of gas gangrene forever with us."

The table on p. 817 from Zeissler and Neller indicates the *incidence* of *anaerobes* in a peace-time series of gas gangrene wounds and in European soil samples.

These anaerobes are characterized by pleomorphism, or the capacity to occur in different forms. This sometimes renders their identification on a morphologic basis very difficult, if not impossible. They vary in size and often present filamentous forms. Usually arranged singly, they may occur in pairs or chains. Their spores may be equatorial or subterminal. The spore-bearing forms are wider than the vegetative or the active bacilli and sometimes are shaped

like spindles, clubs, tennis rackets, or drumsticks.

All members of this group *stain* readily with the usual dyes, although there is great irregularity in the depth of staining. While all are Gram-positive, this reaction sometimes can be demonstrated only when young cultures are examined. In body tissues and in older cultures, they may lose this property rapidly. Some can be decolorized by alcohol much more easily than are the commoner Gram-positive organisms. So-called shadow forms, because of early autolysis, are frequent. Spores are seen as colorless areas if they are mature, or they show as an area of intense staining early in their formation. Occasionally, Cl. welchii can be recognized in tissue fluids by capsule formation.

Anaerobes Present	Occurrence in Twenty-one Gas Gangrene Cases (50 Per Cent Mortality), Percentage	In 200 European Soil Samples: Percentage
Cl. welchii	21 times or 100	100
Cl. novyi (oedematiens)	7 times or 33⅓	45
Cl. oedematis maligni (vibrion septique)	1 time or 5	8
Cl. histolyticum	1 time or 5	2
Cl. tetani	3 times or 13	27

Anaerobes are *cultured* and studied in liquid media, on solid media, and in shake agar tubes. Isolation of single colonies can be very difficult. There are instances where strains described as pure cultures later have been shown to be mixtures of different species. The greatest amount of information is obtained from studying surface colonies on blood agar plates. In this manner, differentiation of certain organisms is facilitated by observing the degree of hemolysis present. The plates must be absolutely dry; otherwise, the spreading tendency of most of the anaerobes renders isolation practically impossible. A much used method of isolating pure strains is picking single colonies from dilution series of shake cultures. Many difficulties are responsible for the impossibility of isolating and identifying clostridia from pathologic material rapidly enough for practical clinical use.

Much of the information about the formation of powerful *exotoxins* secreted into the surrounding media by the clostridia has been learned from the study of clostridia not encountered in gas gangrene—Cl. botulnuim and Cl. tetani. In guinea pigs and mice, the exotoxins produce local gelatinous edema, a varying amount of necrosis and death of the animals. The toxin of C. welchii lyses red blood cells, a property which produces hemoglobinuria, causes focal necroses in the kidney and liver and increases the blood pressure. The toxin of Cl. oedematis maligni caused marked liquefactive necrosis of muscles. The exotoxin is most powerful in Cl. novyi and least powerful in Cl. welchii.

These toxins, which increase the virulence of the anaerobes and make their action more aggressive, stimulate the formation of specific neutralizing antitoxins.

IV. Mechanism of Infection, Toxin Formation and Predisposing Factors

The gravity of gas gangrene is dominated by the peculiar feature that the anaerobes which cause it are not pathogenic in themselves. Unless they are in combination with their toxic metabolic products, or are allowed to form them, they are absolutely harmless. If a culture containing anaerobes is washed carefully and injected into experimental animals, neither gas gangrene nor any other lesion will occur. If, however, these organisms are mixed with a small amount of culture filtrate or with any material containing their toxins in an amount which in itself will not kill the animals, typical gas gangrene, followed by death, occurs.

The exotoxin acts like an aggressin. It allows the anaerobes to multiply in tissues where this otherwise could not take place. In the absence of toxin, the clostridia or

their spores are removed gradually from the site of infection by the processes of lysis and phagocytosis. Experiments indicate that the exotoxin lowers the oxidation concentration, which, in turn, favors anaerobic growth.

In human gas gangrene, toxin practically never is introduced with the organisms and spores. Circumstances must be such that bacilli and spores without toxin will multiply and produce toxin. These favorable conditions prevail when mechanical tissue damage occurs with the formation of necrotic material. Laceration and crushing of tissues, effusions of blood, interference with the circulation, fractures, and foreign bodies produce these conditions. Ultimately, any or all of these predisposing factors can lead to the same disastrous effect—that of creating local conditions in which anaerobes can multiply and produce their toxins. These factors not only facilitate further multiplication and toxin production, but at the same time damage adjacent healthy tissue in such a way that the invading anaerobes can grow in them. In a short time, the distribution of larger amounts of toxin in the body leads to severe systemic changes and finally to death.

The action of anaerobic toxins is not specific. The toxins from one species of anaerobes not only favor multiplication of other toxin-forming anaerobes, but even stimulate the growth of anaerobes which do not themselves form toxins. Aerobes associated with anaerobes in a wound act similarly, by destroying tissues and thus preparing them for the anaerobes. Concurrently, aerobic metabolism lowers the oxygen concentration of the tissues, and thus creates conditions favorable for the anaerobes. Conversely, granulation tissue with its rich blood supply never allows gas-forming anaerobes to produce gas gangrene.

Without a knowledge of the principles which underly anaerobic growth, it is hard to understand why gas gangrene is relatively so rare when the anaerobes which can produce it are so widely distributed. Wound contact with the gas-forming anaerobes certainly cannot be prevented. All efforts, then, must be directed against permitting those conditions to supervene which will allow these organisms to develop, multiply and destroy.

The *varieties of trauma* which predispose to gas gangrene fall into six groups: (1) Crushing and lacerating accidents, particularly compound fractures, where street and farm dirt, rich in anaerobes, gains access to injured tissues, particularly muscles. Frequently a damaged blood supply reduces locally the oxygen concentration and favors anaerobic growth. (2) Gunshot wounds and other puncture wounds into which anaerobes are introduced by contaminated missiles, mud and gravel, and bits of clothing. (3) Injuries to the gluteal and perineal regions where fecal contamination readily occurs. (4) Abortion and instrumental delivery occasionally have caused gas gangrene of the uterus. (5) Hypodermic or intramuscular injections have been complicated by local gas gangrene. (6) Gas gangrene may follow clean surgery, particularly amputation for arteriosclerotic and diabetic gangrene. We have observed four cases after abdominal section. Instances of contaminated catgut are recorded.

In our series of 109 cases the following conditions were complicated by gas gangrene:

Predisposing Conditions	No. of Cases
Compound fractures and lacerations of extremities	55
Amputation of arteriosclerotic and diabetic gangrene	27
Gunshot wounds	7
Operation for bowel obstruction	4
Perineal abscesses	4
Operation for cancer of rectum	3
Amputation for Buerger's disease	5
Gangrenous appendicitis	1
Ludwig's angina	1
Idiopathic septicemia	2
	109

In gas gangrene following hypodermic and intramuscular injections, the anaerobes may be introduced by instruments, or

they may be contained in the fluids injected. An increasingly large amount of data, however, has been collected which indicates that gas gangrene can occur where organisms are not introduced from the outside. A recent possible explanation for that exceedingly important group of gas gangrene cases, where introduction of anaerobes from the outside can be excluded, has gained more and more credence, i.e., the development of the infection from latent spores lying dormant in the tissues. It has been shown conclusively that anaerobic spores can be demonstrated in a high percentage of instances in the organs of healthy animals. These spores probably lie latent in man, especially in the aged. Gas gangrene has been produced experimentally when animals harboring latent spores were injured locally. It seems likely that the trauma from hypodermic injection and that caused by operation may damage tissues containing spores to the degree that conditions are produced which allow the development of gas gangrene.

V. PATHOLOGY

Gas gangrene is essentially a *disease of the muscles*, and this principally because of their high glycogen and glutathione content. The infection begins at the site of injury, and becomes manifest within six to forty-eight hours. The involved muscles swell within their sheaths as the infection spreads up and down. Rarely does gangrene spread transversely to adjoining muscle groups until late in the disease, unless these groups also are traumatized. As the muscle swells, it takes on a brick-red color, becomes lusterless, and loses its contractility. Bubbles of gas appear, and these travel up and down the muscle within the fascial planes and along the vasculoneural bundles. Comparatively soon the muscle substance becomes friable and its color changes to yellow, blue, and finally, black. When the muscle disintegrates, it resembles thick chocolate sauce or broken down liver.

Gas and fluid products of disintegration may escape from the muscle and infiltrate the surrounding tissues, often traveling far beyond the limits of the initially diseased area. The surrounding skin first becomes pale, then tense and ischemic. At this stage, the muscle may take on a creamy tint. The skin turns to a purple hue and finally breaks down into foul, necrotic patches. Lymphatic vessels often are seen as dark streaks produced by the broken-down products of hemoglobin. Blebs filled with liquid and gas are common.

The rapid ischemia and necrosis of muscle is caused by the exotoxin of the anaerobes and by the pressure exerted by the gas formed by these organisms. The muscles are pale and have a boiled appearance before complete disintegration takes place. The appearance of the wound usually is not characteristic because of the mixed infection which often is present.

Microscopically, the muscles show tiny gas bubbles, invasion of anaerobes, separation of fibers, disappearance of the sarcolemma, cloudy swelling of the muscle fibers, and finally a gelatinous disintegration. As a rule, the absence of any inflammatory reaction around the involved tissue is striking.

VI. SYMPTOMS, SIGNS AND CLINICAL DIAGNOSIS

Pain, fever, accelerated pulse rate, and the local changes are the outstanding clinical features early in the disease. The degree of prominence of these signs and symptoms and the order of their appearance is extremely variable.

The *pain* has been characterized as out of all proportion to the injury, and is often the first symptom to attract the attention of the patient and physician. Much importance should be attached to this complaint because local changes are difficult to make out when a cast or massive dressing has been applied. We have seen fulminating gas gangrene supervene in a supposedly clean wound and the patient complain almost not at all. There are so many differences in temperament that the

degree of pain is hard to evaluate. The pain from the causative lesion, as compound fracture or amputation, frequently masks the pain brought on by the anaerobic infection.

So many gradations of *temperature elevations* were encountered that we feel that fever is no reliable indicator of the severity of the infection. One cannot depend on the initial postoperative temperature elevation to indicate the occurrence of infection because most operations carry a postoperative temperature rise. General fatal toxemia may develop without, or with very little, fever. On the other hand, a very high temperature does not justify a hopeless prognosis.

The sudden *elevation of the pulse rate* to 100 or 150, caused by the action of absorbed toxins, may precede the elevation of temperature. The pulse rate is said to be higher proportionately than the fever. In our series a sudden elevation of the pulse rate was the earliest clinical sign in 80 per cent of our cases. Several patients, however, showed no rise in pulse rate. It must be remembered that the pulse rate after the operation frequently is high, and that the increased increment due to the onset of infection may be hard, if not impossible, to visualize. The *odor* from the wound in fairly advanced or full-blown disease is typical. It is extremely heavy, and resembles that of rotten eggs. During the War it was called "the death smell." By some authors it is described as "sickeningly sweet," by others "sourish," and by still others "mousy."

The anaerobic toxins sooner or later produce *constitutional manifestations*, which often are very severe. Stupor, coma, and delirium frequently occur in the severe, and always with the fatal, cases. Rapid destruction of the red blood cells resulting in pronounced jaundice is characteristic of blood stream invasion by the anaerobes. None of our cases with severe jaundice survived. A marked anemia occurs. A *leucocytosis* which may be high (25,000 to 90,000) takes place in infections associated with Cl. welchii. The white blood cell count seldom exceeds 10,000 to 15,000 when Cl. welchii is absent. In our cases the average leucocyte count was 17,000, with 85 per cent polymorphonuclear elements. A secondary anemia was present in 60 per cent of our cases. Fall of blood pressure is frequent and can be very severe.

The *local swelling* rapidly increases with the formation of gas and edema. A *serous secretion* from the wound is an early manifestation. In later stages it becomes hemorrhagic, profuse, turbid and brown, depending largely on the organisms present. The edges of the wound at first are red, then bronze-colored, and finally mottled. The sign which has given the disease its name is the *production of gas in the tissues.* This is demonstrated by crepitation on palpation and a tympanitic sound on percussion. Often the gas is expressed from the wound or from the blebs in bubbles. *X*-rays show gas deep in the muscles early in the disease when it cannot be demonstrated in any other fashion.

We believe the local manifestations to be all important in diagnosis. On that account, we stress again and again the vital importance of keeping under constant supervision all wounds which are potentially infected with anaerobic organisms. Time and time again we have erred by not inspecting and swabbing our wounds early, thus postponing irretrievably the making of the all-important diagnosis.

From a careful and very complete study of the clinical manifestations of all of our cases, made in retrospect, we are forced to conclude that the disease had begun on the average four days before we recognized it. From this admission it must be apparent how important is the stipulation that the local wound must be kept under constant supervision and suspicion. Swelling, reddened wound edges, serous and serosanguinous exudate, and gas bubbles by *x*-ray are the most reliable clinical findings which should lead to smear and culture of the exudate from the depths of the wound.

VII. BACTERIOLOGIC DIAGNOSIS

The specific bacteriologic diagnosis depends entirely upon the available laboratory facilities. To isolate and classify adequately the anaerobes in a given case of gas gangrene may take several weeks or longer, and this only when highly experienced workers with ample facilities for this special work are available. Management of surgical practice demands the essentials of bacteriologic analyses where special facilities are not available. Surgeons not sufficiently experienced in staining and examining smears of suspicious material should consult laboratory workers immediately. Should the Gram stain of a smear from sufficient suitable material show large Gram-positive rods, the possibility of gas gangrene developing should be considered, even in the absence of all symptoms or signs of this infection. If the material is insufficient, a negative smear is not convincing proof of the absence of these organisms. A culture in meat or brain broth may be helpful. The materials examined must be carefully selected. Exudate from infected wounds, fluid from blebs, and pieces of muscles always are satisfactory. Too much emphasis cannot be placed on the stipulation that swabs should be taken from the depth of all suspected wounds. The preparation of the smears at the bedside with a platinum loop is recommended strongly.

In our series, where cultural studies are far above the average, Cl. welchii was reported present in 86 per cent of the cases. The other 14 per cent were reported as large Gram-positive rods.

Only on superficial examination can several conditions be mistaken *differentially* for gas gangrene. Extensive hemorrhage into the tissues can simulate gas gangrene. In this condition the area under consideration is firm to the touch, dull to percussion, but shows no evidence of gas. The patient shows no severe constitutional reaction, and is bacteriologically negative. The use of hydrogen-peroxide as a wound antiseptic sometimes produces temporary crepitation. The absence of other important signs differentiates it from gas bacillus infection. Subcutaneous emphysema from trauma or badly administered hypodermoclysis arouses suspicion.

Next to anaerobe septicemia, gas embolism is the most important complication. Gas bubbles enter the blood stream and cause death from occlusion of small blood vessels in the heart, lungs, or brain.

The *prognosis* in gas gangrene depends mainly upon the duration and severity of the infection and upon the effect of the therapeutic measures instituted. The mortality rate in other reported series of any size varies between 40 and 90 per cent. The death rate in this series was 51 per cent. In our series gas gangrene occurred six times as often in lower extremities as in the upper. Our mortality in lower extremity cases was over 60 per cent, while that in the upper extremity was only 10 per cent.

VIII. TREATMENT

The choice of *prophylactic treatment* presents serious problems. In a wide variety of injuries there is the possibility that gas gangrene may develop. We also know that the incidence of the actual disease really is very low. It is exceedingly difficult, therefore, to lay down specific rules as to when, and in what cases, prophylactic antiserum should or should not be given. The cost of the serum is considerable, and serum reaction and sensitization are unpleasant factors which should not be overlooked. It requires much experience and careful consideration to decide whether the danger of gas gangrene is sufficient to warrant the institution of prophylactic measures. The character of the injury and the degree of contamination are the decisive factors upon which the attending physician must base his decision.

Certainly, in lacerated wounds, compound fractures, and gunshot wounds, débridement is essential. This consists of the mechanical removal from the wound of all devitalized tissue and foreign material.

This operation should be performed as carefully as possible in order to avoid adding injury to already damaged structures, particularly muscles. The wound should be accessible to inspection at all times. At the slightest indication, swabs for smear and culture should be taken, not from the surface suture line, but from the depths of the wound. We have never seen a clean wound infected by this procedure, and we have found frequently that the constant suspicion of the disease, which has led to this practice, has made the diagnosis possible days earlier than it otherwise would have been made. Early culture from the depths of the wound upon the slightest suspicion is the most valuable lesson we have learned from this study. We must never forget that an infection can brew deep in a wound and show little or nothing on the surface or at the suture line.

If a plaster cast must be applied, there should be one or more windows for observation of the wound. *X*-rays to demonstrate gas bubbles, taken immediately after the management of the injury, may be helpful for comparison with films exposed later when gas bacillus infection is suspected.

The theoretical reasons for the *prophylactic administration of antiserum* against gas gangrene are the same as those which actuate the use of antiserum when tetanus is the infection feared. In gas bacillus infection, however, we do not know which of the several organisms present may be the potential incitants of gas gangrene. There is no doubt that a polyvalent serum must be used, the constituents of which depend upon the incidence of the several species of anaerobes which can cause gas gangrene. The number and variety of antisera recommended and accepted by the council of pharmacy is an evidence of the confusion which has arisen as to just what the incidence of these organisms is. These antisera contain different combinations of antitoxins and a varying number of units.

The prophylactic use of antiserum still is experimental. We believe that the so-called prophylactic dosage is too small, for we have seen gas gangrene develop in spite of its administration. We recommend that no amount less than the full therapeutic dose of antiserum should be given prophylactically. One minimum therapeutic dose of a popular antiserum contains:

10,000 units Welch antitoxin
10,000 units oedematis maligni (vibrion septique) antitoxin
200 units novyi (oedematiens) antitoxin
200 units sordelli antitoxin
25 units histolyticus antitoxin

Where gas gangrene seems likely to occur, a second dose of therapeutic serum should be given. An indication for a second dose of serum exists when examination of the wound reveals suspicious organisms. The administration of antiserum for prophylactic purposes should be subcutaneous.

When prophylactic measures have failed, or when, on entry the patient has a well-established infection, more *radical early active measures* must be undertaken. As soon as the onset of symptoms and signs of gas gangrene is recognized, wide incisions in the suspected area should be employed. These release the gas under pressure and allow access of free oxygen to the tissues. These incisions should be made longitudinally in the involved muscle and not pass across fresh uninvolved muscle tissue or across blood vessels which supply the adjoining muscles. Irrigations with Dakin's or Pilcher's solutions or with any other chemical irrigating solution which releases oxygen is recommended. Meleney uses zinc peroxide as a chemical in this connection. This gives off its oxygen very slowly, thus producing a prolonged oxygenation of the tissues.

PILCHER'S QUINOFORMOL SOLUTION

Quinine sulfate	1.00 Gm.
HCl	0.50 c.c.
Glacial acetic acid	5.00 c.c.
NaCl	17.50 Gm.
Formol (40 per cent)	1.00 c.c.
Thymol	0.25 Gm.
Alcohol	15.00 c.c.
Aqua q.s.	1000.00 c.c.

Polyvalent antitoxic serum in therapeutic doses should be administered frequently and in large amounts (100,000 to 150,000 units or even more), intravenously. Injection of the serum around and into the tissues involved should not be made because uninvolved tissues may be damaged by fluid pressure, and conditions favorable to the growth of the anaerobes be established. Injection of oxygen into the affected parts is too dangerous because of the possibility of air embolism. Deep inhalation of oxygen may be helpful. Fluids by mouth should be forced. Hypodermoclysis, infusion, glucose administration and blood transfusions are supportive.

X-ray treatment of gas gangrene has been recommended recently. Thus far, *x*-ray therapy has been so combined with the more established forms of treatment that its specific advantage cannot be evaluated. In isolated instances, the *x*-ray appears to have a measure of efficiency. Until a series of cures is reported in undoubted cases of gas gangrene where *x*-ray alone is the therapeutic agent, no real decision can be made. At present, this therapeutic adjunct is definitely in the experimental stage, but deserves a trial. The considerations concerning *x*-ray treatment apply equally to the use of sulfanilamide and related compounds which have been reported to be helpful clinically as well as experimentally.

If gas gangrene persists after the therapeutic measures noted above have been instituted, *late radical measures*, such as amputation, must be performed. The amputation should be so designed that healthy tissue, especially muscle, should not be damaged, because muscle injury contributes to further spread of gas gangrene. One of us (C.L.C.), has devised a tendoplastic amputation through the femur at the knee which has proven useful in gas gangrene in the lower extremity. The underlying principle of this new operation is that no muscle is sectioned in its fleshy portion. Only the tendinous portions of the thigh muscles are divided at their insertions on the tibia. This operation not only has limited the ascent of gas gangrene in our later cases, but often has been instrumental in preventing its occurrence in arteriosclerotic and diabetic gangrene where amputation through the femur was indicated.

IX. SUMMARY

An attempt has been made to clarify the bacteriologic principles underlying this disease. These principles and the conclusions drawn from the study of a large peacetime series of gas gangrene cases form the basis of this paper. The signs and symptoms are explained from the point of view of altered physiology. Particular attention has been directed to the necessity of making an early diagnosis. The various forms of treatment have been enumerated and a useful amputation for this condition has been described.

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SULFANILAMIDE IN SURGICAL PRACTICE

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SULFANILAMIDE has come quickly to the front rank of our most useful and powerful chemotherapeutic agents. It is particularly valuable in surgery as it lends itself so effectively to the treatment of a wide variety of infections and pathologic conditions. While early use will often obviate surgical interference, it will not always replace the scalpel and sound surgical judgment should temper its use in conjunction with surgical principles. A vast amount of clinical work and factual material born of research is steadily adding to our knowledge of this amazing compound. Although normally possessed of a wide factor of safety, to some individuals it is highly toxic.

Sulfanilamide,[1] para-amino-benzene-sulfonamide, was reported by Long and Bliss[2] before the Southern Medical Association in Baltimore, November 17, 1936. Mellon, Gross and Cooper[3] treated one of their group with it in August 1936 and attribute saving of life to this compound. They remark that it was synthesized by Gelmo in 1908 and has been a compound familiar to organic chemists since; they quote Domagk as stating that Horlein found in 1909 that one of the azo dyes, an allied compound, was effective against hemolytic streptococcal septicemia in mice. They trace the recent history and development of these chemicals, beginning in 1934, with workers in Germany, England and France, and remark that at present some 200 sulfonamide compounds are being investigated.

The variety of trade names has at times led to confusion. Sulfanilamide may be procured under this name or that of prontylin, prontosil album, stramid, sulfamidyl, sulfonamide-P, consulanyde and streptocide (the latter two British). The above preparations of sulfanilamide are readily procurable and discussion in this paper will be limited to this compound. Prontosil, the disodium salt of 4′-sulfamido-phenyl-azo-7-acetylamino-1-hydroxynaphthalene 3:6-disulfonic acid, has been shown by a number of workers to break down, liberate or reduce to P-amino-benzene-sulfonamide; this was corroborated by Bliss and Long.[4] Rapidity of absorption and raising of the sulfanilamide concentration in blood, plasma, spinal fluid, etc., has been shown to take place more rapidly by oral administration than in any other manner.

Safe, effective, proper use of sulfanilamide requires some knowledge of its peculiar characteristics. Symptomatology is unusual and side actions are numerous and vary considerably with the individual. Untoward reactions range from mild disturbances to the most severe manifestations. Idiosyncrasies are often severe, may occur singly or may manifest themselves in combinations. The obvious clinical effectiveness of the drug has caused its widespread use to outrun tedious, detailed, animal experimentation.

CLINICAL APPLICATION

Bliss, Long, and Feinstone[5] have attempted to show the strains of streptococci which yield to sulfanilamide therapy. They call attention to three main classes as grouped by Brown,[6] noting that the strains of alpha group and gamma group have not yet been classified. The subgrouping of Lancefield[7] of beta hemolytic streptococcus into groups A, B, C, D, E, F, G, H, and K, is followed. They found group A responding most readily; groups B and C less so, in order. But apparently much more work remains to be done along this line.

Clinically, beta hemolytic streptococcal infections yield so readily to sulfanilamide that it should be employed as early as a positive, or even presumptive, diagnosis can be made; whether it be a single infecting factor or associated with other organisms. A bacterial diagnosis is desirable, but many of these infections are so characteristic that they can be diagnosed in their early stages without benefit of laboratory. Of this group, erysipelas is an outstanding example, also phlegmonous swellings or cellulitis of the extremities which do not immediately follow the typical course of a staphylococcal infection. Likewise streptococcal infections of the foot, occurring in trichophyte infection with cracks of the epidermis between the toes, or blisters or lesions from mal-fitted shoes or socks, marching, etc., and puncture wounds of the hands, Ludwig's angina, phlebitis, etc. Prompt administration of sulfanilamide in these cases is most effectual, reducing the mortality, materially shortening hospital stay or period of disability, and often averting surgical interference. The resulting scars, contractures or deformities, which all too often occur in the hands and feet from these infections, can be largely prevented by the early and judicious use of sulfanilamide. A recently treated case is typical.

This was an infection of the lower leg, involving the skin and subcutaneous tissue, including the periosteum over the mid-portion of the shin, in a white man of 34. He was admitted with a temperature of 101°F., leucocyte count of 18,500, toxic symptoms, acute cellulitis over the mid-portion of the left shin with exquisite tenderness and severe pain which radiated up the leg. Sulfanilamide therapy was instituted immediately and within twenty-four hours his temperature had reached a normal level. It rose to 99.8°F. on the third day of treatment and following this remained normal. On the fourth day the leucocyte count was 9,400 and shortly after returned to normal. I am quite confident that osteomyelitis was averted.

In several cases of cellulitis of the foot, undoubtedly due to a hemolytic streptococcus, there was a marked drop in the red blood count in an otherwise normal individual, early in each case, before the action of sulfanilamide could have been credited with this effect.

The most striking instance was in a white man, age 39, who was awakened at 5 A.M. by an itching sensation at the base of his right second toe. Rubbing resulted in a sharp, stinging sensation about the area, rapid progress of swelling and severity of pain. He was seen at 9 A.M. on the same day, when his temperature was 103.2°F., leucocyte count 17,300. A slight crack in the epidermis was noted between the first and second toes in a scaly area of trichophyte infection. Half the upper forefoot was covered with a red rash; a streak of lymphangitis extended to the knee. The patient was seen an hour and a half later in the hospital after a complete laboratory check-up. He was then quite toxic, drowsy, suffering violent headache, pains in his back and abdomen, two well defined lines of lymphangitis extended from his foot to his groin, where a noticeable lymphadenitis was present. The forefoot and first three toes were swollen to twice normal size.

The foot was elevated, ice caps were placed on the groin and about the foot for two-hour intervals with an hour of relief. Sulfanilamide in 15 gr. doses, with an equal amount of bicarbonate of soda, was administered by mouth every three hours. Toxic symptoms increased during the next twenty-four hours. The red cell count, which on admission was 5,400,000, dropped to 3,200,000 in the first twenty-four hours. Leucocytes were 16,000 and temperature 102.4°F. on the second day. There was little change in his red cell count on the third day, but leucocytes decreased and at noon the temperature dropped to 100°F. The drug was reduced to 15 gr. every four hours. Swelling in the foot was arrested on the second day, definitely subsiding by noon of the third. The great toe gave evidence of a localizing abscess; however, this subsided rapidly and no surgical interference was necessary. On the fourth day the temperature was normal; the red cell count had dropped to 3,000,000. Sulfanilamide was reduced to 10 gr. at four hour intervals and 300 c.c. of citrated blood was administered, from which the patient suffered a slight reaction. On the fifth day he was given 5 gr. of the drug every four hours,

and it was entirely discontinued on the sixth day. He was allowed to walk with the aid of crutches on the tenth day. He left the hospital on the tenth day after admission in excellent condition, with no swelling about the foot, with a few palpable glands remaining in the left groin, and one area of slight lymphangitis. No swelling or disability of the foot recurred.

Many infections of the hand, of which Kanavel[8] wrote so well, respond to sulfanilamide therapy. The following typical cases are illustrative:

A dentist fell in the dark, driving the rusty end of a quarter-inch bolt into the fleshy pollicis muscles of his right hand a distance of an inch and a quarter. Iodine was applied, followed by dry dressings, and the next morning he presented himself with a temperature of 100°F. and a leucocyte count of 15,000. The entire hand was greatly swollen and extremely painful. He was immediately given tetanus antitoxin and was hospitalized. Hot, moist compresses were applied. Temperature and symptoms increased throughout the day and night, but on the following night the wound began to discharge a grumous red-brown pus from which beta hemolytic streptococcus and staphylococcus aureus were cultured. The infection gradually subsided; he was discharged from the hospital a week after admission, but the symptoms immediately recurred with an ointment dressing. He was then given 5 gr. of sulfanilamide every three hours. The infection promptly subsided, the wound healed and remained healed.

A striking case occured in a white man, age 23, a gardener, who punctured the fourth finger of his right hand with a thorn, near the knuckle. Twelve hours later he was aware of a swollen red area. Twenty-four hours after the injury his temperature was 99.8°F., leucocytes 20,000; the hand was greatly swollen, extremely painful and he was unable to move wrist or fingers. He appeared quite toxic, and was admitted to the hospital. The right hand and forearm were placed in a continuous hot tub; narcotics were administered. A complete blood picture was obtained which was essentially normal aside from the high leucocyte count and corresponding increase in neutrophiles.

Sulfanilamide therapy was immediately instituted, 10 gr. every four hours with an equal quantity of bicarbonate of soda. The following day infection had not advanced. On the third day there was definite improvement. The infection quickly localized, and on the sixth day fluctuation was noted over the knuckle. This was incised under ethyl chloride and a great amount of pus and necrotic material was expelled. This produced a luxuriant growth of beta hemolytic streptococci and hemolytic staphylococci. The following day the fourth finger distal to the knuckle was incised and further pus and necrotic material obtained. The wound quickly healed and the patient returned to work, without disability, in less than three weeks from date of injury.

This localizing influence of sulfanilamide I have observed in two other cases and it was noted also by Mellon, Gross and Cooper[3] in a case of Ludwig's angina; also by Colebrook and Kenny.[9]

I have advised its use in two cases of Ludwig's angina. One case did not yield to this therapy but finally localized and culture revealed staphylococcus aureus. The other case subsided without incision. Fulghum[45] reports a similar experience.

Long and Bliss[10] have treated erysipelas and there are other more recent reports. Mellon, Gross and Cooper[3] have collected 1,000 cases of erysipelas treated by sulfanilamide or similar compounds with a mortality of less than 1 per cent. I have treated two such cases of very ill patients with prompt relief and subsiding of signs and symptoms within a week, although both patients were rather debilitated for another two or three weeks.

Of five cases of phlebitis, which I have treated with sulfanilamide, four made rapid recoveries but one showed marked idiosyncrasy for the drug and it had to be stopped immediately. This case ran the usual prolonged course.

Although acute rheumatic fever has not yielded to sulfanilamide therapy and the ordinary rheumatoid arthritis cases have failed to show improvement, I have noted complete relief of symptoms in acute arthritis following sore throat and the so-called "grippal" attacks.

One patient presented himself with an exquisitely painful, tender, completely disabled right shoulder, which came on a few days after a mild sore throat had subsided. Aspiration of the subdeltoid bursa and shoulder joint yielded no fluid. Leucocyte count was 10,000, temperature 99.6°F. The administration of 10 gr. of sulfanilamide and an equal quantity of bicarbonate of soda, over a period of two days, brought about complete recovery.

Another patient, who was being treated for an attack of grippe, was referred for orthopedic care when he developed cellulitis of the forearms and a very acute arthritis of the cervical spine, right knee and left ankle. His temperature was 101°F., leucocyte count 13,000. These symptoms rapidly subsided under sulfanilamide therapy, 60 gr. per twenty-four hours, in equal doses given at four-hour intervals, with an equal amount of bicarbonate of soda. However, symptoms promptly recurred after the drug was discontinued. The therapy was repeated only to have recurrence when again discontinued. His blood sulfanilamide was raised to 8 mg. per 100 c.c. a third time and two teeth with apical abscesses were removed and tonsillectomy was done. He was quite cyanotic before operation and there was some apprehension regarding a general anesthetic. However, nitrous oxide and oxygen induction followed by ether with oxygen was tolerated without difficulty although his color throughout the operation was ghastly and remained so for several hours after.

He made a speedy and complete recovery, in spite of a localizing process below the left ankle which healed promptly following incision. Culture revealed beta hemolytic streptococcus; the pus was a watery, straw-colored fluid.

This patient had been treated a year before for an acute exacerbation of an old osteomyelitis (of twelve years' duration), with a draining cavity below his left knee which was ankylosed. On the basis of his statement that the original infection was streptococcal, sulfanilamide therapy was used and, to my amazement, all of the swelling and inflammation subsided within forty-eight hours. I have treated other cases of streptococcal osteomyelitis with sulfanilamide but have found it essential to establish drainage, remove sequestra, and carry out the usual surgical procedures before healing occurs.

Otitis and mastoiditis are frequent surgical problems and I have seen both, in the acute stages, yield to sulfanilamide therapy, some even subsiding without surgical interference. I have seen no chronic cases of otitis media treated with this drug. Smith, Mayer, Agerty, Fowler and Layton[11] report a case of otitis complicated by streptococcal meningitis yielding to this form of treatment.

Mellon, Gross and Cooper[3] have found twenty-nine cases of streptococcal meningitis in the literature, of which twenty-five have recovered with sulfanilamide administration. While not a direct surgical problem, it is sometimes a complication.

They discuss puerperal sepsis, streptococcal septicemia, peritonitis, pneumonia, cystitis, pyelitis, tonsillitis, pharyngitis—all of which may be complicating factors to the surgeon—also streptococcal empyema. They cite seventeen cases reported in the literature, and Brown[12] reported two cases cured by intrapleural injections of prontosil.

Mellon, Gross and Cooper,[3] in summarizing these various manifestations of streptococcal infections, remark that streptococcal meningitis, with a previous 100 per cent mortality, has now a death rate of 15 per cent; they consider it quite remarkable that, often under oral administration of the drug, the spinal fluid promptly becomes sterile. They comment on the uniformly favorable responses in the lesser manifestations, such as lymphangitis, tonsillitis, cystitis, pyelitis, mastoiditis, etc.

Collins,[47] noting streptococci in stool cultures and rectal scrapings in chronic ulcerative colitis cases, gave sulfanilamide a therapeutic trial and obtained an initial favorable response in eight cases out of eleven. Bannick, Brown and Foster[48] noted excellent results in more than 50 per cent of twelve cases treated. Ward[49] makes similar observations.

I have used sulfanilamide in hemolytic streptococcal gangrene of the skin, of which Christopher[13] writes. The first case was in an old man who developed gangrenous

areas on his right leg following contusions sustained in an automobile accident. These areas later sloughed away and healed nicely under warm saline compresses. Two other cases responded as well. I also advised sulfanilamide in a case* of gangrenous slough starting about the ulcerated gum margin of a tooth, which spread about the mouth and over the lower lip and chin, a process of two weeks' duration.

This patient was extremely ill, with temperature of 101.4°F., pulse 120, respiration 24, blood pressure of 88/48 (patient previously had suffered with hypertension). There was a gangrenous ulcer on the calf of right leg. Anasarca was present. Urine showed 2 plus albumin with an occasional red blood cell and frequent granular casts. Leucocyte count was 32,000, hemoglobin 65 per cent, Wassermann negative. Non-protein nitrogen was 48. Because of her critical condition, the patient was given an initial dose of 80 gr. of sulfanilamide by nasal tube with 40 gr. of bicarbonate of soda. This raised the blood level of sulfanilamide to 4.3 mg. per 100 c.c. in six hours. Following this, 20 gr. were administered with an equal amount of bicarbonate of soda in the same manner every four hours for three days, followed by 10 gr. three times a day. On the second day of treatment, the patient received 0.03 Gm. of neoarsphenamine by intravenous route, also a transfusion of 350 c.c. of citrated blood. On the third day 300 c.c. of blood was given and 275 c.c. on the fifth. Sulfanilamide was discontinued after the eighth day, its maximum concentration in the blood reaching 27.3 mg. per cent on the fourth day of treatment. The red cell count never dropped below 3,000,000, which occurred on the second day of treatment, when leucocytes reached 72,000—undoubtedly the result of sulfanilamide action. Dr. Owings remarked that it was astounding to see the patient's greatly swollen face recede and toxic symptoms subside almost immediately. The extension of the gangrene stopped at once, the sloughing lip and a portion of chin was removed and the patient appeared to be doing very well when separating slough caused a carotid branch to rupture and she died suddenly on the fifteenth day following onset of treatment.

* Case treated by Dr. James C. Owings.

I have seen cases of sinusitis yield promptly to this therapy, the first being a personal observation. After being awakened with a throbbing, severe pain in the left side of my face, I endured several hours of sleeplessness; a presumptive diagnosis of sinusitis of the left antrum seemed logical. The discomfort was so great that I ingested 10 gr. of sulfanilamide with an equal quantity of bicarbonate of soda every three hours. I had taken 20 gr. when, early in the morning, I consulted a laryngologist who found an acute sinusitis of the left antrum. Washings developed a pure growth of beta hemolytic streptococcus. The infection subsided within thirty-six hours without sequelae of any kind.

Rowe[14] reports recovery of brain abscess treated with sulfanilamide and drainage.

A number of other infections of interest to the general or specializing surgeon are of importance. Brown and Bannick[15] find sulfanilamide of value in treating infections of the cervix associated with carcinoma, and state that they have been able to employ radium therapy when clinical experience previously had indicated that it was not feasible because of the danger of exacerbation of infection. They also cite a case of acute infectious mononucleosis, associated with an acute sore throat, in which recovery seemed to be materially hastened by use of this drug.

Long and Bliss[10] treated a case of acute meningococcal pyarthrosis of the knee occurring in a six weeks old baby, in which the knee aspirations became sterile within three days and prompt recovery resulted. Sulfanilamide was given in the feeding formula.

Keeper and Spink[16] treated gonococcal arthritis with sulfanilamide and found that infected synovial fluid became sterile within two days in two of the patients (one was found to be sterile before treatment).

Colston, Dees and Harrill[17] report ten cases of gonococcal arthritis treated in a similar manner, and remark that "in several cases orthopedic splinting was necessary to prevent deformity resulting

from the infection, although tenderness and fluid had entirely disappeared." Other cases have been reported. However, I have treated several cases and believe that splinting is rarely indicated. Sulfanilamide therapy should be combined with aspiration and air injection of the joint (Bohlman[18]) whenever effusion occurs. Air injections have marked therapeutic value in acute gonococcal arthritis, and air serves to prevent adhesions, adds to comfort, minimizes distention of the joint capsule and recurrence of fluid. I believe that 15 or 20 per cent of gonococcal infections will not yield to sulfanilamide therapy.

Pneumococcus occasionally causes pyarthrosis and these cases may well be treated with sulfanilamide, particularly if Types I, II and III are involved. Rosenthal[19] and co-workers[20,21,22] report experimental work with mice, showing its effectiveness on these strains. Mellon and co-workers[3] report cases of pneumococcal pneumonia, type III, responding very favorably to this therapy.

Occasionally an abscess occurs with brucellosis to plague the surgeon. Francis,[23] Richardson,[24] Stern and Blake,[25] Toome and Jenkins,[26] Loyd[27] and others report favorable results in undulant fever treated with this drug.

Urologists Young and Vest[28] report two cases of almost moribund individuals, suffering with lactus aerogenes septicemia, cured in a remarkably short time with sulfanilamide therapy. They give an interesting table of results in 109 consecutive cases, in which 168 types of organisms were found before treatment and sixty-three after treatment, with reduced growth in nineteen. (Table I.)

Cokkinis[29] notes surprising improvement after sulfanilamide therapy in such unrelated conditions as acute cholecystitis, throat infections, appendiceal abscess, suppurative arthritis, infections of the urinary tract, subphrenic abscess, septic compound fractures, and diffuse peritonitis. I have used it in acute tenosynovitis and bursitis. In traumatic surgery, sulfanilamide is of particular value. I have used it with excellent results in compound fractures, infected hematoma (streptococcus), and in lacerated or perforated wounds. In a number of cases, mixed infections of staphylococcus and streptococcus were found.

TABLE I
ORGANISMS ENCOUNTERED IN 109 CONSECUTIVE CASES

	Before Treatment	After Treatment	Reduced Growth
Escherichia	37	9	4
Aerobacter	39	16	2
Citrobacter	1	0	0
B. coli (undetermined)	1	0	0
Proteus	19	9	1
Pseudomona group (pyocyaneus, etc.)	6	2	1
Salmonella	1	..	1
Dysenterae (Flexner, Sonne, Dispar)	3	2	1
Friedlander's	1	0	
Alcaligenes	2	0	
Hemophilus	1	0	0
Staphylococcus aureus	22	8	6
Staphylococcus albus	18	5	2
Coccus (undetermined)	1	1	
Streptococcus alpha	16	4	1
Streptococcus beta	2	1	0
Streptococcus gamma	6	5	0
	168	63	19

In gas gangrene I have found it an effective, almost specific, agent. Although I first used it[30] in an apparently hopeless case of gas gangrene simply with the hope of stopping symbiotic growth of streptococcus and lessening toxicity, to my amazement it accomplished a rapid and complete recovery. Following this, Long and Bliss[10] found that experimental infections of B. welchii in mice yielded more quickly and readily to this therapy than did the streptococcal infections. This is quite in contrast to the work of Spray[31] who made a study of the effect of sulfanilamide compounds on various sporulating anaerobes in vitro. He found that B. welchii and the various proteolytic types of Clostridium were scarcely affected by sulfanilamide. However, various authors have remarked on discrepancies

of behavior of this drug in vitro and in vivo, and subsequent cases have proved the clinical value of this therapy in gas gangrene when used early and combined with adequate early surgical measures.

Sulfanilamide therapy must be combined with immediate measures for relief of pressure and tension in the affected part. It takes slight pressure to obliterate capillary circulation, and capillary activity is absolutely essential in cell and sera reaction to an invading organism; also to carry sulfanilamide into the affected areas. The drug will not save a limb where poor surgical technique or judgment has ruined circulation. Dressings or supports must in no way impede capillary or venous circulation. In a wound or a compound fracture where Clostridium is present, a slight amount of gas will add greatly to the tension. With circulation impeded, tremendous multiplication of the bacilli will occur in tissues deprived of capillary circulation; this bacterial growth takes place so rapidly that, in appropriately stained microscopic sections of these tissues, vast numbers of these organisms will be found without cellular response of the tissues. Open wounds should be debrided when necessary and left open. The injury may be severe enough to warrant incisions for relief of tension; this should be done at the first sign of failure of capillary circulation in any area. Amputation will be necessary where trauma has too severely injured the circulatory mechanism of a part or extensive thrombosis has occurred.

I have seen two cases of failure with sulfanilamide therapy where gas was present about the wound. The first was in a very old man with a strangulated hernia, in which an emergency operation was imperative. His blood sugar was very high, he had not been under treatment preceding the operation, and following it his temperature continued elevated. He developed gas in the tissue about the wound, but no gas bacilli were cultured or found. Colon bacillus, strain undetermined, was cultured from the wound and from the blood stream just before death. The patient was a very poor operative risk. I suspect the gas was due to a colon bacillus. The second case was a diabetic also, and therapy was started about twenty-four hours after onset of gas gangrene. B. welchii was cultured and was present in great numbers in smears from the wound. Here, mass action of bacteria created severe or fatal toxicity before an effective concentration of sulfanilamide could be obtained in blood and body fluids.

I have never seen a diabetic recover from a well developed case of gas gangrene, regardless of therapy employed.

I have found three cases of gas gangrene which developed after sulfanilamide was given routinely in prophylactic doses following severe injuries. The first was a 5 year old white boy with a severe compound fracture of the right lower leg in which immediate débridement was done, some fragments of tibia were removed, and a vitallium metal plate was used to fix the tibial fragments. Complete closure was done, resulting in much tension, and a parallel incision was made over the calf. After operation it was noted that pulsation in the dorsalis pedis was not palpable, although pulsation was evident in the popliteal space. Sulfanilamide therapy, 5 gr. every four hours, was instituted when the patient had recovered sufficiently to swallow following operation. The next day his temperature was elevated, and when cyanosis appeared in the toes, the plaster splint was split and later removed. He became very toxic, with a temperature of 104°F., and both lower extremities became cyanotic. Cultures of the wound revealed B. welchii and incisions were made. Later, amputation at the midthigh, was done twenty-eight hours after admission. B. welchii was cultured from the stump wound, as was staphylococcus albus. Sulfanilamide therapy in the above dosage was continued for four days although no total amount was recorded and no blood concentration determinations were done. The patient was given several transfusions, also received intravenous glucose and saline

several times. Sulfanilamide may have reached a sufficient concentration in the blood and tissues following the second operation to prevent extension of gas bacillus in the stump; it was inadequate at first and the wound should have been left wide open and dressed loosely and lightly. The fracture might have been temporarily immobilized with sand-bag trough.

The second case was a severe arm injury in which swelling and thrombosis caused infection to outrun sulfanilamide concentration in the damaged part and amputation was necessary because of damaged circulation. A third case, multiple gun shot wounds of the lower leg with fracture, behaved in the same manner. Ample incisions of skin and fascia must be done very early—preferably débridement with wounds left open and lightly packed with dressings.

Chronic streptococcal infections have been reported as yielding to this therapy. In my experience it has occasionally been helpful, but it has been completely disappointing in the pyocyaneus infections, the chronic staphylococcal infections of bone and in some cases of streptococcal infections. In a case of bursitis over the trochanter with draining sinuses and recurrent attacks of fever, there was a mixed culture of beta hemolytic streptococcus, staphylococcus aureus, pyocyaneus, and proteus. This yielded readily during exacerbations, with a temperature drop to normal, on sulfanilamide therapy. Eventually, however, it was necessary to excise the infected area completely and section the iliotibial band.

ADMINISTRATION

Sulfanilamide should not be given by intravenous route; it is too toxic. The method of choice is by mouth. If the patient is unable to swallow, and a nasal tube can be passed, a solution or suspension may be placed in the stomach by this means. Hypodermocylysis may be employed and the drug administered subcutaneously. Intrathecal administration has been employed, but I am inclined to class this with intravenous administration. Intrapleural injections have been utilized, also rectal administration and topical applications. I have found little value in dressing wounds with this compound; likewise the application of compresses containing sulfanilamide has been disappointing.

ABSORPTION

Oral administration is simple and easy, and given in this manner sulfanilamide is absorbed readily. Marshall, Emerson and Cutting[32] have found that maximum blood concentration after ingestion of sulfanilamide occurs within three to five hours—as rapidly as when it is administered subcutaneously. Marshall and coworkers[33] found that the concentration of sulfanilamide was essentially the same in various tissues, except in bone and fat in which it was lower; and remark that simultaneous determination of sulfanilamide in blood and spinal fluid of man over a period of several hours' administration, reveals the concentration in the spinal fluid to parallel that of the blood but at a somewhat lower level. I have found the concentration in a joint effusion to be three times as great as that in the blood. Mellon, Gross and Cooper[3] advise the subcutaneous route in persistent nausea or in cases where oral administration has failed to raise the blood level sufficiently. Long and Bliss[34] suggest the intrathecal route in streptococcal infections of the meninges. Brown[12] reports two cases of streptococcal empyemia treated by intrapleural injections of prontosil.

DOSAGE

Amount of drug and frequency of dosage should be governed by the severity of infection, and the size as well as the age of the individual. Very ill patients with severe infection, and very large patients, should receive a heavier initial dose with subsequent ones of sufficient size and frequency to maintain the proper blood concentration of sulfanilamide. The younger patients, and those with milder infections, should be

given correspondingly smaller initial doses, although in treating gonorrhea, Vest[28] is inclined to give a large initial dose. The dosage may be varied, depending on the blood level, and should be decreased as soon as clinical improvement is noted. In cases showing marked toxicity or idiosyncrasy to the drug, it should be discontinued immediately. This will be discussed later.

Because of marked individual variation in tolerance to the drug, I have avoided the large or massive initial doses except in extremely ill patients, preferring to start with 10 to 20 gr. repeated every three or four hours. Even very ill patients with beta hemolytic streptococcal infections and some of those suffering with gas gangrene have responded to this method of administration in a remarkably short time. This, I believe, is the safer course except in severe infections.

If an initial massive dose is desired, that suggested by Long and Bliss in various papers and recently emphasized by these authors[35] may be used. In adults, depending on size and age, 45 to 75 gr. is the initial oral dose, followed in six to eight hours by 15 to 20 gr. which may be repeated every four hours. In children of 50 to 100 pounds, 30 to 45 gr. forms the initial dose, followed in six or eight hours by 5 to 15 gr. Children weighing 25 to 50 pounds, the first dose is 20 to 30 gr., followed by 5 or more gr. in six or eight hours, repeated every four hours.

If, under this method, the blood concentration is not sufficiently high, subcutaneous injection of the drug may be carried out. For parenteral therapy, 700 c.c. of 0.8 per cent sulfanilamide solution can be given subcutaneously, followed by 500 c.c. every eight hours for the first twenty-four hours. In children of the first group, 300 to 500 c.c. may be given as the initial dose, followed by 200 to 300 c.c. every eight hours; and children of second group—100 to 200 c.c. followed by 100 to 200 c.c. every eight hours.

In infants, 15 gr. per 10 pounds during the first twenty-four hours may be used and Long and Bliss suggest continuing treatment until a definite clinical effect is obtained, then gradually decreasing by one-third of the original amount and continuing at this level until convalescence is established. These authors suggest smaller dosages where prophylaxis is indicated. In preparing the parenteral solution, Long[36] suggests boiling physiologic saline solution until sterile, then 0.8 Gm. of the chemical is added per 100 c.c.; the solution is again boiled for several minutes, and the volume may be made up with the addition of sterile saline solution; I have found this quite important as the sulfanilamide is soluble up to 1 per cent and crystallizes very readily if this point is reached. The solution should be kept hot until needed, then cooled to 98.6°F., and this temperature maintained while it is being given. If it is placed in an ice box, crystallization will take place. I believe that fever is more apt to occur with parenteral administration than when the drug is given by mouth; this was noted in several cases where both methods were used alternately.

CONCENTRATION OF SULFANILAMIDE IN THE BLOOD

Improvement in the patient's condition and the level of sulfanilamide in the blood are the two most important factors in governing continued dosage. Marshall[37] has given a method for determination of sulfanilamide in the blood and urine. Fuller[38] and Proom[39] likewise give detailed methods of determining sulfanilamide concentration in blood or other body fluids. A small compact outfit with which determinations can be made in fifteen or twenty minutes is now available on the market.*

Blood concentration will vary to some extent in different individuals on similar dosage. It is apparently higher in proportion to dosage in patients suffering with chronic glomerulonephritis. Marshall[40] has shown in dogs that sulfanilamide clearance

* LaMotte Chemical Products Company, Baltimore, Maryland.

is 20 to 30 per cent of the simultaneously determined creatinine clearance, and notes that clearance of the drug is independent of plasma level and its excretion is increased by rate of flow of the urine. I have noted acute glomerulonephritis associated with streptococcal infections which improved under sulfanilamide therapy with proper blood-level maintenance.

Condition of the kidneys should be determined before a massive initial dose of sulfanilamide is given.

Blood concentration may vary from a trace to 20 or 28 mg. per cent. Long and Bliss[10] advise reducing the dosage when blood level of sulfanilamide reaches 15 to 20 mg. per 100 c.c. Brown and Bannick[15] have noted efficiency of lower concentrations of sulfanilamide and this parallels the author's experience. Long and his co-workers advocate a blood concentration in the neighborhood of 10 mg. per 100 c.c. The highest blood concentration I have seen was 27.3 mg. per cent which occurred on the second day of therapy, following an initial dose of 80 gr. given by nasal tube in a patient afflicted with chronic nephritis. Long[57] notes a maximum of 54 mg. per cent and has had one case maintained on 10 mg. per cent or above constantly for 365 days (presumptive cure of bacterial endocarditis). The majority of my cases have responded with a concentration of 4 to 8 mg. per cent. Marshall and co-workers[32] have shown that maximum concentration occurs in four to six hours. It is advisable to do a determination after an initial mass dose at the end of five hours and a second determination in twelve to fifteen hours with subsequent checks daily, preferably twice a day as long as the therapy is continued.

The more resistant infections should have a sustained, higher blood-level concentration.

EXCRETION

Sulfanilamide is excreted by the kidneys, and therefore, as noted above in Marshall's work, the fluid intake should be limited. Long and Bliss[41] have found that higher concentrations of sulfanilamide in the urine are either bacteriostatic or bactericidal and they believe that these levels are necessary; they are more readily obtained with the urine volume kept relatively low. Large doses must be given daily, and the blood concentration must be high to maintain the urine concentration. They caution that because of toxicity, the drug should be used in urinary tract infections only after conservative treatment has failed and they suggest constant observation for the early recognition of toxic signs. Marshall[40] has studied the mechanism of excretion in experimental animals, comparing it with the accepted creatinine clearance measure of glomerular filtration and he finds that 70 to 80 per cent of filtered sulfanilamide is reabsorbed in passage along the tubules.

Adair, Hesseltine and Hac[42] have found sulfanilamide in cervical secretion and menstrual fluid in quantities small enough to be of questionable bacteriostatic effect. They note that it is excreted in milk, both in the free form and as acetyl derivatives, and continues to be excreted in milk after blood level is "negligibly low." They found that with doses of 30 to 60 gr., the total amount excreted was never more than 1½ per cent of the amount administered, being noted seventy-two hours after discontinuance of medication. They observed that sulfanilamide was transmitted to the placenta and fetus in rabbits, causing marked increase in mortality of the young; and also that it has been found in human placenta and cord blood. They urge "utmost caution" in administration during pregnancy and lactation, even suggesting that breast feeding be discontinued while it is being given. Pratt, in discussing this paper, feels that an insufficient amount of sulfanilamide is excreted in the milk to be dangerous to suckling infants.

Farrell, Lyman and Youman[43] obtained prostatic fluid of dogs after intravenous administration of sulfanilamide and tested the samples with B. coli and staphylococcus aureus and noted bactericidal activity. They

consider 10 to 15 mg. per cent in the urine to be adequate antiseptic concentration. This is in contrast to the work of Long and Bliss.[41] Clark and Branham[44] note that younger individuals excrete sulfanilamide more rapidly than do older people. Marshall, Emerson and Cutting[46] have noted a temporary decrease in renal function following large doses.

TOXICITY

"The median lethal dose of sulfanilamide for man is unknown and it is impossible to deduce it from animal experiments." So remark Marshall and co-workers,[46] Stafford[50] states that in a series of fifteen cases treated on the surgical service in the Johns Hopkins Hospital during the past six months, three have been irrational, with one man wildly disoriented. The latter leaped from a window, fracturing his femur and sustaining other injuries from which he died three days later with complications of anemia and uremia. He was being treated for a cellulitis of the hand. This man had received a total of 85½ gr. in the space of thirty hours; his hemoglobin dropped from 100 to 50 per cent in the first forty-eight hours following initial dose.

The literature abounds with reports of clinical manifestations of toxic symptoms and signs. These consist of cyanosis, sometimes acidosis with air hunger; anemia and leucopenia, which may be of an acute hemolytic type or a more slowly progressive disturbance; nausea, epigastric pain, precordial pain, dermatitis, photophobia, elevation of temperature, headache, vertigo, tinnitus and deafness, jaundice, porphyrinuria and hemoglobinuria, loss of taste for tobacco and alcohol, weakness, lassitude, anorexia, abdominal distress, diarrhea, mental disturbances which may consist of hilarity and excitement, moroseness, drowsiness, depression, disorientation, etc. Death may occur as a result of one or more disturbances associated with ingestion of sulfanilamide where the dosage has been too long sustained or where there is individual susceptibility to toxic effects of the drug.

The mechanism of cyanosis is not yet clear. Marshall and Walzl[51] studied the blood of seven patients, comparing the oxygen-carrying capacity with the total iron pigment of the blood. They concluded that clinical cyanosis may be unaccompanied by any decrease in oxygen-carrying capacity and may occur without the presence of non-functional iron pigment. However, in two cases the apparently non-functional iron pigment was present. They believed it to be methemoglobin. They remark that sulphemoglobin may also result following injection of sulfanilamide but conclude that these two pigments probably do not, in the main, explain the the cyanosis so frequently encountered. Discombe[52] sounds the sharpest warning on the continued administration of sulfanilamide when cyanosis is severe and associated with anemia. He states that deeper cyanosis cannot occur after hemoglobin drops to 30 per cent and continued medication will convert more hemoglobin into the inert form. King and Allen,[53] after examining oxygen saturation of arterial blood in cyanotic cases, concluded that in no instance was oxygen saturation at a point low enough to explain the relatively marked cyanosis. They concluded that it did not play a significant rôle in cyanosis of this type and they note that administration of oxygen failed to relieve this cyanosis. Mull and Smith,[54] on the other hand, found actual lowering of the capacity of the blood for oxygen in one case and advised the use of oxygen and transfusion.

Mellon and co-workers[3] state that a definite hazard exists in unfavorable collateral reactions—"some of which, if not carefully watched and controlled, may become as serious as the primary disease." They list ten fatalities in approximately 4,000 cases.

In a series of forty-five cases, the author has noted an initial rise in red and white blood cells, and a corresponding rise in hemoglobin during the first period of

administration, which averaged three days. Exception is noted of two cases which developed an acute hemolytic anemia immediately. Following the initial rise there was a drop of red and white cells and hemoglobin which continued as much as a week after administration of the drug was discontinued. In one case of acute hemolytic anemia the hemoglobin and red blood count dropped steadily in spite of large transfusions every second day, but ceased shortly after the medication was stopped and returned to normal four weeks later. Another patient, in a space of twelve hours, showed a drop in red blood cells of 1,500,000 with a drop in white blood corpuscles to 4,000. He exhibited a dermatitis, peripheral neuritis, and photophobia at the end of this time, although only 40 gr. of the drug had been ingested in divided doses given at four-hour intervals.

Long and Bliss[34] speak of granulocytopenia with depression of myeloid elements; in some cases, an associated angina. Bigler, Clifton and Werner[55] found that sulfanilamide caused a depression of white blood cells, even to leucopenia. They noted no agranulocytosis or granulocytopenia with this depression. In a few examinations of platelets no change was noted. Kracke[56] found agranulocytosis in two cases with one death. He remarked on the proclivity of sulfanilamide for causing a severe type of acute hemolytic anemia with rapid hemolyzing of the red blood cells of from one half to one million per day. He also remarked on the tendency of some cases to show stimulation of leucocytes which was sometimes preceded by moderate depression. He has observed six patients in whom the leucocyte count ran as high as 74,000, noting that marked agranulocytic shift to the left occurred. I have seen several patients develop a high leucocyte count in a similar manner. In studying protocols, a number of cases have appeared in which I believe death was due to sulfanilamide and not to the diagnosed disease. Long[57] estimates that between 1,100 and 1,200 house patients have been given the drug in Johns Hopkins Hospital with only one death due to sulfanilamide, which was noted above.

Wageman and Blake[58] noted nitrogen retention in three cases. In two it subsided promptly with discontinuance of the drug. They remark that one patient had no known preëxisting kidney disease, while another had acute nephritis when treatment was started and died later of uremia. One of their patients developed toxic hepatitis and they received one with thrombocytopenic purpura who had been taking salol and phenacetin in combination. They believe this may have caused his blood dyscrasia.

Davis[59] administered sulfanilamide to rats by subcutaneously injecting a suspension of the drug in distilled water. The rats immediately developed a thirst but adjusted themselves in about a week. Microscopic examination revealed amorphous crystals of the drug at the site of injection lying within foreign body giant cell type of reaction. These crystals were found in the spleen and liver, particularly in the capsules of these organs, with occasional "curious proliferations of the endothelium of the hepatic vessels." Occasionally they were found in meninges and in each instance a proliferation of fibroblasts surrounding the crystals occurred.

Mental symptoms may vary from the mildest excitement or confusion types to violent disturbances. The author noted difficulty in driving a car while taking a moderate dosage; and has previously[18] warned against driving any sort of vehicle or flying while taking this drug. This applies also to people working about machinery or where clear perception and rapid reaction is a necessity.

Skin patch tests for sensitivity have not been satisfactory in my experience.

Mellon and co-workers[3] advise carefully selecting a product of a reputable manufacturer to avoid toxicity from impurities.

The dry crystals are the safest form to procure for parenteral use, and the tablets for oral administration. One needs only to

mention the regrettable Massengill elixir[72] with some seventy-six recorded deaths, and Geiling and co-worker's[60] beautiful pharmacologic studies, to warn against untried preparations.

I have not found recorded, or observed, any permanent damage following the use of sulfanilamide. Toxicity varies with the dose and the individual sensitivity. Acute hemolytic anemia, hyperpyrexia and abnormally high leucocyte count, severe mental disturbances, toxic amblyopia, severe neuritis, or other violent manifestations of sensitivity or poisoning are all causes for discontinuing the drug at once. Reuter[67] observes that the result of the treatment is "almost predictable" by the degree of disagreeable manifestations. The more severe, the better the result. Smith[68] speaks of a similar experience.

COMBATING TOXICITY

When toxic symptoms are severe, the drug should be discontinued. For lesser manifestations, the dosage may be decreased. Forcing fluids by mouth, and the intravenous administration of 5 per cent glucose in normal saline will flush the drug out of the body as the rate of renal excretion is stepped up. If acidosis develops, ⅙ molar sodium lactate solution should be given subcutaneously or intravenously as so often advised by Perrin Long. Regular administration of proper quantities of an alkali, bicarbonate of soda, with each dose of sulfanilamide will go far toward preventing acidosis. In case of anemia, a transfusion should be given as frequently as necessary to maintain a reasonable level of hemoglobin and circulating red blood cells.

Vest, Harrill and Colston[61] advise the forcing of fluids to prevent possible crystallization of acid hematin in the renal tubules. Exposure to sunlight should be avoided by patients who develop a dermatitis. Mellon and co-workers[3] note that in some cases the drug caused the rash to spread like a sunburn over areas not exposed to the sun. The same authors caution against the use of phenacetin, acetanilid, methylacetanilid and derivatives of nitrobenzene. These authors feel that not only magnesium sulfate but any cathartic producing liquid stools will increase hydrogen sulfide and will accelerate the formation of sulphemoglobin. They suggest the use of mineral oil and enemata.

These authors also advise the use of vitamins and certain hormones to reinforce the action of sulfanilamide. I have employed vitamin therapy on a wide scale during the past ten years and have noted marked improvement of infections in many cases, with parenteral administration of vitamins B and C.

PRECAUTIONS

Sulfanilamide should be administered only under the closest supervision, preferably in a hospital. Tarbell[62] reports severe illness in an ambulatory patient who disregarded directions concerning the dosage. Long[57] reports three out-patient subjects admitted to the Johns Hopkins Hospital, two suffering with agranulocytosis and one with acute hemolytic anemia, all of whom died. For some time, I have practically limited administration to hospital cases. Before sulfanilamide therapy is begun, red and white blood cell counts, as well as hemoglobin determination and a differential count, should be done. Examination of the urine should be carried out. A determination of non-protein nitrogen should be done and fluid intake and output chart kept. If a massive initial dose of sulfanilamide is given, a blood concentration determination should be made within five or six hours and repeated once or twice daily thereafter. Daily red and white blood cell and differential counts should be done, as well as frequent urine examinations and, where necessary, sulfanilamide concentration determined. Frequent determinations of non-protein nitrogen are advisable. Fluids should be restricted at the onset of therapy and forced the moment severe toxic symptoms become evident. Where

large doses of sulfanilamide are to be employed, blood grouping should be done before administration and, if possible, donors matched so that transfusion can be done on short notice. It is well to bear in mind that in some instances a hemolytic streptococcus will affect the hemoglobin and red blood cell count materially and this should not be confused with possible toxic effects of the drug. It is particularly necessary, during therapy, to observe temperature and leucocyte count as there may be no drop in the febrile reaction as the infection subsides and the toxic action of sulfanilamide causes it to rise or stay elevated. Clinical symptoms and leucocyte count should be the differentiating factors in these cases.

PROPHYLACTIC USE OF SULFANILAMIDE

Long and the author[30] suggested the prophylactic use of sulfanilamide in severe crushing injuries or compound fractures and this is being carried out by many workers. Whiting[63] has administered it to twelve patients, some of whom developed staphylococcal infections and two of whom developed gas gangrene. Study of these cases convinces one that the failure was due to early disturbance or occlusion of circulation. Cobey[64] lists some twenty cases in which sulfanilamide has been used in this manner with one case of gas gangrene resulting, which on review was definitely due to almost immediate occlusion of circulation. The continued administration of sulfanilamide in this case undoubtedly was of value, as B. Welchii was cultured from the amputation wound. Vest and Harrill[65] found that sulfanilamide was of no value in preventing infection following operation for hypospadias. Gaudin, Zide and Thompson[66] found the prophylactic use in transurethral resection did not prevent postoperative infection of the wounds. It is hard to evaluate prophylactic effectiveness, and employment in this manner should be governed by the condition of the individual and the probable risk of infection.

MECHANISM OF ACTION

This intriguing subject is gradually being explained, although much present information borders on the theoretical. Mellon and co-workers[3] call attention to the large amount of sulfanilamide necessary to kill organisms in test tubes, "while smaller, almost infinitesimal fractions are effective in the body." They cite the quantitative aspects of the work of Colebrook, Buttle and O'Meara,[69] who found that a light inoculum would yield to a high dilution of sulfanilamide while a heavy inoculum required an immensely stronger concentration of drug. This corresponds to my observations in the treatment of gas gangrene, where after a certain time the multiplication of bacteria approaches mass action and becomes overwhelming regardless of treatment. Mellon and co-workers believe that "action of the drug is either mediated or reinforced by the host tissues" and they discuss various hypotheses, including the rôle of leucocytes, bacteriostasis and the interference with capsule formation, also the phenomenon of potentiation. They do not regard neutralizing of toxins as probable. They conclude that sulfanilamide increases the mobilization of phagocytic cells at the site of infection with hemolytic streptococci, that the drug has an unquestionable bacteriostatic effect in vivo, and their observations indicate that virulent streptococci may be dissociated into avirulent forms by sulfanilamide. They cite a case in which cultures revealed a pure growth of streptococcus hemolyticus which, after sulfanilamide treatment, yielded a culture of diphtheroids; they comment on the reversibility of diphtheroids to streptococci.

Osgood and Brownlee,[70] on the other hand, believe that sulfanilamide neutralizes or destroys toxins of beta hemolytic streptococcus, that it causes a decrease in the rate of multiplication and assists bactericidal action of human serum and phagocytic action of neutrophiles and monocytes of the marrow and blood, that

it is effective in a concentration of 1:100,000, which is much less than the 1:10,000 now in clinical use. Meyer[71] summarizes his experimental tests on the action of sulfanilamide in animals infected with streptococci with an opinion that the drug effects the bacteria through attenuation of virulence, detoxication and bacteriostasis, with simultaneous coöperation of the body's protective powers.

CONCLUSION

Sulfanilamide, a remarkable and extremely valuable chemotherapeutic agent, is widely known and used. It lends itself to the treatment of innumerable infections arising from many kinds of bacteria. Cheap, easily obtained and administered, first producing notable cures in hemolytic streptococcal infections from which much publicity arose, it inevitably came to trial in all sorts of infections and conditions. Some of these results have been gratifying, others more disappointing. Disasters of minor grade interspersed with more serious disturbances and fatalities have accompanied this rapidly mounting usage. Too often administration has not been tempered by full knowledge of side actions and toxicity. Clinical use proceeded more rapidly than laboratory experimentation. At present the many fascinating phases of sulfanilamide are intriguing a host of laboratory workers and clinicians are recording their observations.

Each month brings information regarding the puzzling behavior of this drug with its diametric effects in different individuals. Eventually all indications and contraindications will resolve themselves into a set of rules. This paper is intended as a present clinical directive.

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Débridement of the bone ends [in compound fractures] usually prevents the death of bone and the resulting formation of sequestra.

All the fillers in this issue were taken from "Experience in the Management of Fractures and Dislocations Based on an Analysis of 4,390 Cases" by the staff of the Fracture Service, Massachusetts General Hospital, edited by Philip D. Wilson (Lippincott).

DISABILITY EVALUATION

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DISABILITY is a term which implies a depreciation of normal ability to perform the functions of established physical accomplishments. It may result from injury, disease or malformation. The period of its existence may be temporary, progressive or permanent. A suitable measuring rod for disability is an important medical subject because there are only two agencies which determine the amounts an injured person shall receive for permanent damage to his body; one is the law-making body which enacts certain specific provisions from which to make awards; the other is the regularly licensed physician to fix the cause and extent of injury. Heretofore in only a few states, by statute, could the court order a physical examination of an injured person. Now under the 1938 rules a physical examination can be ordered in any federal court of the United States.* There is no escape from the responsibility. The subject is destined to arise in the curriculum of medical knowledge and duty.

The nature and extent of any physical injury is always a question of fact. Lay witnesses cannot and are not permitted to describe and give opinion to such facts. The most a layman can do is to describe what he saw and heard. Necessarily, therefore, all judicial inquiries in respect to compensation or damages for injuries result in the use of the expert witness, namely, the regularly licensed physician. His statements and opinions are highly respected for their professional sincerity and intellectual dependability.

In his testimony the doctor, of course, attempts to handle the case as he would in private practice. He thoroughly examines the patient and reports his exact findings from a scientific point of view. He is to disregard the liability for the injury or the influence of legal, political or welfare pressure that may be, and usually is, brought to bear.

The remarkable and discouraging feature about the average hearing in court or before a board, is that an equal number of medical witnesses may appear on either side of a case, one group aiming to augment the significance of the injury, the other to minimize it. The legal profession acknowledges this system, which has been in use for several hundred years. The medical profession cannot remedy such a well established system of judicial procedure. However, out of respect for the scientific background of medical practice and the professional attitude toward search for the truth, it seems that the conduct of the doctor as an expert witness could be greatly improved upon. When called as medical experts on opposite sides of a case in court, the doctors should be able to consult, comparing their physical findings and diagnostic opinions with the same seriousness of scientific intent as in any case occurring in the regular practice of medicine. In this way any oversight could be corrected and differences understood before courtroom proceedings begin. Thus fully informed of both the affirmative and the negative findings, the medical expert, no matter on which side he happened to be called, could have all the available information, as would the medical

* Rule 35 of rules of civic procedure for U. S. District Courts adopted by the Supreme Court pursuant to act of Congress reads: "In an action in which the mental or physical condition of a party is in controversy the court in which the action is pending may order him to submit to a physical or mental examination by a physician."

experts and lawyers on the opposing side. His answers to the questions propounded would thus be more clearly interpreted and his opinions of greater value.

Definite methods of arriving at conclusions as to the evaluation of disability would also greatly eliminate many medical inconsistencies in court. The writer has already fully gone on record in this respect.* The subject is one that should be developed in every medical school as a part of the foundation of medical practice, to give the medical witness a feeling of confidence rather than one of helplessness when facing a court. The responsibility of answering the legal inquiries is even greater than that of participation in a medical consultation. The court often insists upon definite answers to medical questions that are at best controvertible, hypothetical and equivocal. Fundamental training on how to meet such exigencies and how to present medical subjects publicly to non-medical inquisitors would prepare a ground of independent court consciousness in the field of medicine.

When the treatment of an injury has reached a point where the injured person can be dismissed, there are still several stages through which the case must pass from the standpoint of disability.

1. Is the person able to return to some kind of occupational work? If so, his "healing period" has ended.

2. Is he able to do some kinds of work, but not capable of carrying on the same work as could be done previous to injury? If he is limited, then he is partially disabled.

3. If he is partially disabled, is this a temporary partial or a permanent partial state?

4. If he will never be able to do any kind of gainful work, then he is totally permanently disabled.

When the extent of permanent disability is to be determined the analysis must include three phases of the subject, as follows:

1. The mechanical alterations resulting from anatomic and physiologic changes such as a limited range of motion, shortening, deformity or other pathology must be thoroughly brought to light.

2. Compensatory and adaptive possibilities of rehabilitation and adjustment to new environment must be considered. Change of vocation, occupation or other application of working ability may be of importance.

3. The extent of function as influenced by the physical changes ultimately becomes the medical basis upon which to determine the working capacity and any award for permanent disability.

The mechanical alterations are revealed by clinical examination. The rehabilitation, reëducation and social readjustments of a permanently disabled person are important but not entirely within the scope of the physician to control or fully determine. Much depends on the mental capacity, the willingness and ingenuity of the affected person and the social opportunities available to him.

The extent of function of a disabled body or part of the body is determined by judging the ability of the altered body to perform the activities and duties of physical exertion. By using function as a basis, the disability evaluation may be expressed in percentage when the test is applied to ordinary manual labor, to a specific occupation, to a particular person or to any set of circumstances relative to the person's ability to perform.

For several years the writer has been advocating a standard of measurement based upon the percentage value of the various factors constituting function. The essence of this method is as follows:

Seven basic factors are selected which make possible such activities of the employed workman as lifting, pulling, bending, turning, pushing, walking, jumping, running, grasping, throwing, kicking and stepping. These factors are:

* McBride, E. D. Disability Evaluation: Principles of Treatment of Compensable Injuries. Philadelphia, J. B. Lippincott Company.

1. Quickness of action.
2. Coördination of movement.
3. Strength.
4. Security.
5. Endurance.
6. Safety as a workman.
7. Prestige of normal physique in securing or pursuing employment.

It is reasonable that if each of these functional factors enters into estimating the extent of disability of the injured workman, then each should bear its relation to the evaluated loss unless any single factor, alone, is responsible for total disability. For instance, if the disabled member of the body is estimated to have lost 50 per cent in strength it does not necessarily follow that the other factors are affected to the same degree. It is necessary, therefore, that each of the factors constituting normal function should be given an evaluation according to its relative importance to the working capacity of the individual. The value of each of these factors may be expressed in percentages as follows:

	Per Cent
1. Quickness of action	10
2. Coördination of movement	20
3. Strength	20
4. Security	10
5. Endurance	20
6. Safety as a workman	10
7. Prestige of normal physique	10
Total	100

These respective values may be adjusted or other factors added, but the total cannot be more than 100 per cent. When evaluating the disability of a member of the body, the percentage of loss of each functional factor is determined and the result expressed in terms of disability. For instance, if it is found that there has been a loss of 25 per cent in the activity factor, then 25 per cent of 10 (value of the factor) will result in 2.5 per cent disability. If it is decided that there is an interference of coördination of movement to the extent of 50 per cent, then 50 per cent of 20 gives 10 per cent disability. Applying the test to each of the factors in the 100 per cent scale and summing up the resulting percentage of loss to each factor, one arrives at the total representing the amount of permanent disability.

Let us take for example the case of an elbow, which due to injury has left the motion of flexion and extension limited to an arc from full extension to 90 degrees flexion. This would be an anatomic loss of 50 per cent motion but not necessarily 50 per cent loss of function. The examining physician might come to the conclusion that the loss in each of the seven functional factors is as follows:

1. A delayed action of 25 per cent.	
25 per cent of 10 (value of quick motion factor).	2.5
2. An awkwardness and loss of coördination of 25 per cent.	
25 per cent of 20 (value of coördination factor).	5.0
3. A weakness of 15 per cent.	
15 per cent of 20 (value of strength factor)	3.0
4. An insecurity of 15 per cent.	
15 per cent of 10 (value of security factor)	1.5
5. A diminished endurance of 15 per cent.	
15 per cent of 20 (value of endurance factor)	3.0
6. An increased risk of 10 per cent.	
10 per cent of 10 (value of safety factor)	1.0
7. An averse influence to employment of 15 per cent.	
15 per cent of 10 (value of physique factor)	1.5
This would give a percentage disability to the arm of	17.5

Where there are multiple lesions to the body or to a limb the combined percentage of loss to the part as a whole must be expressed. In such cases it is necessary to determine the percentage of loss from each individual disability and to compute the relative value to the part as a whole. It would not be mathematically correct simply to add the percentages of loss from multiple lesions, because their sum might be more than 100 per cent. The loss to the part as a whole must be determined through relation of each disability to 100 per cent. For instance, an angulation deformity, as the result of a fracture, may contribute a certain percentage of the disability of a limb, while ankylosis of a joint may also contribute a certain per-

centage. These percentages would not be added, but each would be discounted as a proportional depreciation of 100 per cent. For example, suppose that in addition to the partial ankylosis of the elbow and disability of 17.5 per cent as above cited, the examination reveals a fracture of the humerus, which has healed in angulation of 20 degrees, leaving a partial loss of use of the arm that is permanent. The percentage of loss from the fracture deformity alone as it contributes to each functional factor might be estimated as follows:

	Per Cent		
Delayed action	15	15 per cent of 10 (value of quickness)	1.5
Awkwardness	20	20 per cent of 20 (value of coördination)	4.0
Weakness	10	10 per cent of 20 (value of strength)	2.0
Insecurity	15	15 per cent of 10 (value of security)	1.5
Diminished endurance	10	10 per cent of 20 (value of endurance)	2.0
Increased risk	15	15 per cent of 10 (value of safety)	1.5
Averse influence to employment	10	10 per cent of 10 (value physique)	1.0
The percentage of disability to the arm due to the fracture would be			13.5

In evaluating the loss to the entire arm resulting from the disability of ankylosis in the elbow and the disability of fracture of the humerus, we would not add the 17.5 per cent disability resulting from the ankylosis and the 13.5 per cent resulting from the fracture. On the contrary, the loss from each disability should be discounted as a proportion to 100 per cent i.e., each disability has only a relative share in the disability as a whole. The loss of 17.5 per cent resulting from the ankylosis then would be deducted from 100 per cent and the 13.5 per cent loss due to the fracture would be a percentage of the balance. The results obtained would then be added to find the total percentage of loss. The problem would be stated as follows:

100 per cent less 17.5 is 82.5, balance good arm.
13.5 per cent of 82.5 is 11.1 per cent.
17.5 plus 11.1 is 28.6 per cent.
The disability to the arm then is 28.6 per cent.

As another example we might have three or more disability losses to one extremity, which if added would total more than 100 per cent, as for instance, a partial ulnar nerve paralysis, an ankylosis of the thumb, and a severe ankylosis of the elbow. After evaluating the loss of function resulting from each injury it might be concluded that:

1. The ulnar nerve disability alone would rate a 60 per cent loss to the arm.
2. The ankylosis of the thumb alone would rate a loss of 10 per cent to the arm.
3. The ankylosis of the elbow alone would rate a loss of 50 per cent to the arm.

If added, these three sums would be 120 per cent. An equitable way to relate multiple losses in this instance would be as follows:

60 per cent is loss from nerve injury.
100 per cent minus 60 is 40 per cent—balance good arm.
10 per cent is loss from thumb injury.
10 per cent of 40 is 4: relative amount of loss from thumb injury.
40 minus 4 is 36 per cent—balance good arm.
50 per cent is loss from elbow injury.
50 per cent of 36 is 18: relative amount of loss from elbow injury.
60 per cent plus 4 per cent plus 18 per cent equals 82 per cent.
The partial loss to the arm is 82 per cent.

It is often requested that disability of fingers or toes be expressed in terms of loss of the hand or the foot as a whole. The specific awards provided by law for amputation of the hand or fingers are somewhat as follows:

	Weeks	
Hand as a whole		200 weeks
Thumb	60	
Index finger	35	
Middle finger	30	
Ring finger	20	
Little finger	15	
Total	160	
Hand exclusive of fingers	40	
Total	200	

We might have an injury in which the disability to the fingers and thumb would be as follows:

		Weeks
Loss to the thumb.	25 per cent: 25 per cent of 60 is	15
Loss to index finger	20 per cent: 20 per cent of 35 is	7
Loss to ring finger..	10 per cent: 10 per cent of 20 is	2
Loss to hand exclusive of fingers....	20 per cent: 20 per cent of 40 is	8
		32

Expressed in percentage 32 weeks is 16 per cent of 200 weeks.
The loss to the hand, therefore, is 16 per cent.

These examples illustrate how the doctor can standardize his opinions when evaluating disability in terms of percentage. Even then his judgment may be disputed or his opinions disliked, but his manner of reasoning will be uniform and his final decision worthy of respect.

SUMMARY

The medical expert is an indispensable factor when there is litigation over personal injury. These cases are usually tried in the courts with medical men on both sides. The object of their testimony is to assist in the cause of justice. The doctor will do well to treat the case, in his court testimony, with the same professional attitude as in his private practice. The conduct and scientific testimony of the doctor in court could be greatly improved upon if the medical experts on opposite sides of the case could arrange a consultation, previous to the hearing, for the comparison of their physical findings and diagnostic opinions, similar to the methods of the regular practice of medicine.

There is a need for standardization in the field of disability evaluation. Fundamental knowledge should be made available to the medical student. At present the knowledge of court testimony and opinions required of doctors is acquired to a very great extent after entering medical practice. Most graduates of Grade A medical schools have similar ability. Wide differences of opinion expressed on opposite sides in court reflect a lack of uniform scientific training in industrial surgery and medical jurisprudence.

When a case is tried before an industrial board the terms of the law create a necessity on the part of the Court to ask of the testifying doctor what in his opinion is the percentage of disability. Answers to such a question bring about expressions of opinion in which the percentages of disability are at wide variation.

An attempt has been made to formulate a method of reasoning and establish a measuring rod, in which a more uniform analysis of the case and opinions of percentage of permanent disability can be more scientifically expressed by the testifying doctor.

A measuring rod based on evaluation of function of the crippled part of the body is offered at least as a partial solution to this difficult problem. Examples are given briefly for illustration.

SHORT WAVE DIATHERMY IN INDUSTRIAL REHABILITATION

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IN the treatment of those disabled in industry short wave diathermy has to a certain extent been overemphasized in the past few years. It should be remembered that, in the light of present-day investigations, the only effects obtainable from applications of short wave diathermy are those produced by fairly deep heating of the tissue. While one should not become over-enthusiastic concerning the value of short wave diathermy, on the other hand one should not minimize the extreme value of deep local heating in the treatment of certain types of industrial injuries.

According to Sir John Simon,[29] measures, whether voluntary or compulsory, for the prevention of industrial disease and ill health depend primarily on the knowledge, skill, and coöperation of the medical profession. I have previously pointed out that the chief sphere of usefulness of physical therapy in industrial injuries lies in the treatment of the large group of patients who have suffered from trauma to bones, joints, muscles, nerves, or skin, as well as in the hastening of restoration of articular function.[20] The proper application of short wave diathermy by the skilled surgeon is one of the most important means of hastening rehabilitation in cases of industrial injuries.

DEFINITIONS

Short wave diathermy may be defined as the therapeutic heating of the bodily tissues by means of an oscillating electrical current of extremely high frequency.[21] Industrial medicine has been said to deal with "the rehabilitation of diseased and injured persons in an effort to insure them a livelihood."[28]

A confusing variety of terms has been used to describe the treatment of the bodily tissues by means of short radio waves which vary in frequency from 10,000,000 cycles per second and a 30 meter wavelength to 100,000,000 cycles per second and a 3 meter wavelength. Among the terms which have been used may be mentioned "short wave therapy" and "ultra-short* wave therapy,"[25] "radiothermy,"[1] "radiathermy,"[16] "parathermy,"[33] "neodiathermy,"[17] "short diathermy," and "ultra-diathermy,*"[24] "ultra-short* wave diathermy" and "short wave diathermy."[19] Although there is merit in a number of these designations, the term "short wave diathermy" is now most frequently used in the United States and seems most acceptable, since the waves are short only in relation to the longer conventional diathermy radiations and since they may probably be best considered as another form of diathermy. The Council on Physical Therapy of the American Medical Association considers "short wave diathermy" acceptable terminology.

APPARATUS

The apparatus used for the production of short wave diathermy currents resembles in construction a short wave radio transmitter, such as is used for amateur broadcasting or for police calls, with the exception that the electrical energy, instead of being dispersed from the antennae as in broadcasting, is chiefly confined either between condenser plates producing an electrostatic field, or within a coil producing an electromagnetic field. That portion

* If the wave length is less than 12 meters, the "ultra" prefix is used.

of the patient's body which is within such a field acts partly as a conductor and partly as a dielector, and the tissues are allegedly heated by the production of ionic oscillations and by molecular friction. Power losses occur within the bodily tissues. The entire phenomenon, therefore, is customarily designated as "hysteresis."

A simple simile to illustrate the manner of heating the bodily tissues by short wave diathermy has been suggested.[6] The rubbing of a rubber band with the fingers will produce heating of it by surface friction and this may be compared to the heating produced by an electric current flowing through a substance which offers ohmic resistance. On the other hand, if one holds the rubber band at the ends and stretches it back and forth, heat will be produced by molecular friction inside the band. This latter effect may be compared to that produced when short wave diathermy fields produce molecular friction and heat losses within the bodily tissues. Because of this deep molecular friction, deep heating effects may be produced.

Two types of short wave diathermy apparatus are now commonly in use: the vacuum tube machine (which is preferred by most physicians) and the spark gap device.[18] A third type, the so-called low output generator, has also been described[34] and has been recommended for the application of "low intensity treatment." This last named device produces no obvious heating effects and, since practically all the beneficial effects obtained with short wave diathermy have been attributed by leading authorities only to heating, Taylor[31] seems correct in stating that the so-called low energy apparatus can have "no effect other than a psychologic one."

To be effective, a short wave diathermy apparatus should have a fairly high output in watts. Physicians would do well to purchase only such machines as have been accepted by the Council on Physical Therapy of the American Medical Association.

From the clinical standpoint there is no difference in the physiologic effects produced by short wave diathermy or by conventional diathermy apparatus, but most observers agree that short wave diathermy produces deeper, more uniform heating of various bodily structures and that it is more readily applied, although dosage control is more difficult than with conventional diathermy.

SELECTIVE HEATING

It has often been claimed that short wave diathermy would produce selective heating effects within the human body; that is, that by selecting a short wave diathermy machine which produced short radio waves in the proper wavelength, it would be possible to heat certain bodily structures more than others. While it is true that selective heat production can readily be demonstrated in inorganic substances and dead tissues, nevertheless in the living animal or human being, no selective heating effects can be expected. Careful studies have shown that because of the dissipation of heat by the very efficient circulation of the living animal, no selective heating is produced.[3] In April 1937, the Council on Physical Therapy[5] agreed that, so far as competent investigators have been able to determine, there is no demonstrable selective thermal action produced by short wave diathermy when it is applied to the living body.

THERMOPENETRATION

A number of investigators have concluded that, for various physiologic reasons, short wave diathermy should produce a more uniform and deeper penetration of heat than conventional diathermy; however, a number of clinical observations have failed to confirm this view. In all probability there is a slightly more uniform and slightly deeper penetration of heat with short wave diathermy than with conventional diathermy.

WAVELENGTH

One of the problems which frequently confronts the industrial surgeon who is

considering the purchase of a short wave diathermy machine is that of selecting one with the proper wavelength. Each manufacturer's representative who interviews him extols the virtues of the particular wavelength which his own apparatus happens to produce. Much controversy has ensued concerning the value of various wavelengths between 3 and 30 meters. Careful investigations on rats have indicated no particular differences in certain effects on experimental tumors whether the wavelengths employed were 3.4 meters, 6 meters, or longer.[14] Likewise, actual experiment with fourteen different short wave diathermy machines of wavelengths between 6 and 25 meters revealed that, for heating purposes, there appeared to be no particular advantage of any one wavelength over another.[23] Furthermore, in pelvic heating experiments with various wavelengths from 9 to 24 meters, no significant differences in heating were observed.[3,15] In addition, studies on the heating of live human muscle and fat revealed no significant heating differences with the use of various wavelengths between 6 and 24 meters.[2,4]

It would seem to be the definite consensus that the heating effects of various wavelengths between 3 and 30 meters are approximately the same and that the surgeon who contemplates purchasing a short wave diathermy apparatus may disregard this problem of wavelength and select his apparatus by basing his consideration on other factors.

BACTERIOLOGIC EFFECTS

It has been claimed that short wave diathermy will produce certain specific effects on bacteria and their toxins;[22,27,30] however, other studies[7,10,12,13] indicate quite definitely that short wave diathermy produces no specific bactericidal effects other than those attributable to the heat produced. Aside from the effects of prolonged high fevers produced by short wave diathermy upon the gonococcus (*Neisseria gonorrhoeae*) and the meningococcus (*Neisseria meningitidis*), both of which have an extremely low thermal death time, there is no evidence that short wave diathermy will have any specific effect upon bacterial infections in the living human body.

PHYSIOLOGIC EFFECTS

I have discussed this problem fully elsewhere;[21] suffice it to say here that it seems quite definitely to be the consensus that, in light of present observations, no specific physiologic effects other than those attributable to heating have been proved to be produced by short wave diathermy. To date, insufficient studies have been made to warrant drawing absolute, final conclusions, and one therefore cannot deny the remote possibility of the existence of such specific effects.

DISADVANTAGES OF SHORT WAVE DIATHERMY

Unless a careful technique is followed, there is a possibility of producing electrical burns which heal rather slowly; these burns, however, are less likely to occur than is the case when conventional diathermy is used.

Probably the greatest disadvantage in using short wave diathermy is that there has not yet been developed a simple method of measuring dosage accurately. One must depend entirely upon the sensation of the patient in order to obtain the proper amount of heat. For this reason, such methods must be used with extreme caution in the treatment of patients with anesthetic areas of the skin, young children, or individuals who are unconscious.

ADVANTAGES

It is generally agreed that short wave diathermy produces more homogeneous, deeper heating of bodily tissues than do other therapeutic methods of applying heat, and that there is greater facility of application than with conventional diathermy.

INDICATIONS

Although the use of short wave diathermy has been much exploited, nevertheless it must be considered favorably in the treatment of many types of industrial injuries, particularly those in which deep heating of the tissues is desired. With the foregoing explanation of the effects and limitations of the method, the industrial surgeon may proceed to the consideration of those diseases or injuries in which short wave diathermy may be of value. It should be kept in mind that in many instances simpler methods of applying heat may prove almost equally satisfactory, and it is only when deep heating of the tissues is indicated that short wave diathermy need be used. It has been shown[2] that the temperature deep down in the quadriceps muscle of the thigh may often be raised as much as 5 or 6°F. (about 2°C.) within a half hour by means of the application of short wave diathermy. It is not possible to produce such rises of temperature in the deeper tissues by any of the simpler methods of therapeutic heating. Applications of radiant heat from an infra-red or luminous heater, or applications of conductive heat from hot solutions or hot pads, never produce penetration of heat to a depth of more than a centimeter or two.

Conclusions concerning the therapeutic efficacy of any physiologic or medicinal agent must be based on the evaluation of the procedure as applied to living patients. The following are conditions commonly seen in industrial surgery in which the use of short wave diathermy may prove to be a valuable adjunct to other therapeutic measures:

Arthritis, Traumatic. The use of short wave diathermy may frequently serve to hasten resorption of local inflammation in traumatic arthritis, and it may be used in conjunction with massage and in some instances with slow rhythmic movements to hasten restoration of function. The applications should usually be of low intensity for the first few times, and the intensity may then be gradually increased.

Bursitis. A first attack of bursitis may often be relieved by the use of short wave diathermy applied for thirty minutes daily and followed by massage and relaxed motion. At first, the massage must be very light until the acute inflammation has subsided and no motion should be started until the pain has been somewhat relieved. Still later, active exercise should be prescribed. It is believed by some[11,32] that calcified regions may be caused to be absorbed by the prolonged use of diathermy. It is certain that in such cases careful localization of the heat from a diathermy machine frequently gives much symptomatic relief.

Contusions. For the first twenty-four hours after injury heat should not be applied owing to the danger of increasing the amount of extravasation of blood into the tissues. Cold applications and tight bandaging are indicated. Thereafter, diathermy may sometimes be used with considerable success in promoting absorption and relieving swelling. Massage is usually applied in conjunction with the applications of diathermy.

Dislocations. Following reduction of a dislocation, if a removable plaster splint is used, daily applications of heat, sometimes in the form of short wave diathermy, followed by massage may be used. Restoration of function will frequently be hastened by following such a program and a more rapid return of function can be expected with this procedure than with the common practice of strapping with adhesive plaster alone.

Myositis and Fibrositis. In acute inflammations of the muscles or fibrous tissues the application of deep heat by means of short wave diathermy may frequently be of great benefit. Such conditions as "lumbago," "arthralgia," "muscular rheumatism," "pleurodynia," "torticollis," and "fibrositic or indurative headache" are due to inflammation of either muscular or fibrous tissues. In fibrositis, very firm

massage should follow the application of heat whereas, in myositis, the massage should be deep but less firm.

Neuritis. In the various neuritides, applications of heat may diminish pain and inflammation. When the involved nerves are deeply situated, the use of short wave diathermy is the most effective means of producing the required increase in local temperature. During the acute stage the intensity of the heat should be moderate; as the treatments continue, the amount of heat may be increased.

Fractures. In the treatment of fractures one should consider not only the anatomic result, but also the functional result. In this type of industrial injury the use of physical measures can play a very important part in rapid restoration of function. The use of heat, massage and mobilization at the earliest possible moment should be considered. These measures tend to increase circulation, prevent adhesions and improve muscle tone. Heat, which may sometimes be applied in the form of short wave diathermy, serves mainly as an adjunct to massage and exercise.

Sprains and Strains. Strains are usually treated in the same manner as contusions, and heat should not be applied during the first twenty-four to forty-eight hours. Thereafter it should be used in gradually increasing doses. With a sprain there are lacerations of blood vessels, muscles, nerves, synovial membranes and tendons. There is usually hemorrhage accompanied by swelling and muscle spasm. After the first forty-eight hours, during which immobilization, firm support and applications of cold are usually in order, diathermy may be started. This is usually continued daily, and is followed as a rule by massage, later by relaxed movements, and finally by active exercises.

Suppurations. There is considerable controversy at present concerning the value of short wave diathermy in the treatment of suppurative lesions. For the most part those who have claimed that it is of great value in such treatment have not presented carefully controlled studies to prove their contentions. Nevertheless, a considerable number of investigators[8–10,26,35,36] have observed beneficial results. There is sufficient evidence for one to be justified in considering the use of short wave diathermy in the treatment of furuncles, carbuncles and other superficial suppurative lesions. To date, however, insufficient comparative studies have been made for one to determine definitely whether or not short wave diathermy is more effective than other simpler forms of mild heating.

SUMMARY AND CONCLUSIONS

Short wave diathermy, properly applied, may frequently prove valuable in the treatment of certain industrial injuries. By producing deep local heating, it may help to hasten restoration of function following trauma. The most satisfactory short wave diathermy machines are the vacuum tube devices of high wattage output which have been approved by the Council on Physical Therapy of the American Medical Association.

Because of the rapid dissipation of heat by the efficient circulation of blood, no selective heating effects are produced in the human body by short wave diathermy. The heating effects of various wavelengths between 3 and 30 meters are approximately the same and the physician need not be overly concerned about selecting an apparatus which produces any particular wavelength within this range.

To date, short wave diathermy has not been shown to produce any bactericidal or physiologic effects other than those attributable to the heat which it generates, and no accurate clinical method of measuring dosage of short wave diathermy has yet been developed.

Short wave diathermy produces more homogeneous, deeper heating of bodily tissues than do other therapeutic methods of applying heat. Because of this deeper heating effect, short wave diathermy can be used in preference to simpler heating

methods as an adjunct in the treatment of deep lying inflammation, trauma and other types of industrial lesions.

Short wave diathermy can frequently be used to advantage to aid the industrial surgeon in his chief aim: restoring the patient to his occupation in the shortest possible time.

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BOOK REVIEWS

ANUS, RECTUM, SIGMOID COLON. DIAGNOSIS AND TREATMENT. By Harry Elliott Bacon, B.S., M.D., F.A.C.S., F.A.P.S. Introduction by W. Wayne Babcock, M.D. Foreword by J. P. Lockhart-Mummery, F.R.C.S. Philadelphia, 1938. J. B. Lippincott Company. Price $8.50.

The diseases of this portion of the intestinal canal have come to occupy an important part of medicine. The science and art of proctology have made such giant strides during recent years that a textbook which offers in systematic form this science and art has a definite place in our literature.

The author in his preface says, in part, "It is felt the resulting volume, which has consumed over seven years of constant notation, elimination and construction based on private practice, clinical experience in two of the largest proctologic departments in the city, and post-graduate studies abroad . . . is sufficiently detailed to serve as a text for the student and a reference book for the general practitioner . . . For the general surgeon it is informative on surgical procedures applicable to proctologic syndromes. . . . " Which, after all, is a book review, and inasmuch as we concur in the above statements, little more is left to be said.

Naturally, the field is covered most thoroughly. The text is well written. The illustrations are excellent. An extensive bibliography is included at the end of each chapter. Also, there are indices to authors and subjects covered. The book is 855 pages long and is to be recommended most highly.

SPINAL ANESTHESIA. By Louis H. Maxson, M.D., with a foreword by W. Wayne Babcock, M.D. Philadelphia, 1938. J. B. Lippincott Company.

Since spinal anesthesia is now universally used, since tragic deaths sometimes occur shortly after the anesthetizing agent has been introduced, and since various postanesthetic complications are encountered, Dr. Maxson's book is likely to prove of great value to those who employ this method of anesthesia.

Dr. Babcock in the Foreword says, "The dangers of spinal anesthesia lie with the user more than the drug. Thus, we would insist that the user be skilled and well trained, that he wear sterile gloves and be as meticulous in aspesis as though he were operating upon a joint or the spinal cord itself. . . . If the contents of the dura are not wounded, if impure solutions are not used, then the great majority of postanesthetic complications attributed to spinal anesthesia are absent; most deaths from spinal anesthesia could have been prevented. . . . Early, before we learned how to select patients, to watch them after the injection and especially how to accomplish resuscitation, one patient in 600 died on the operating table. Now with a well trained attentive personnel and resuscitative appliances instantly available, we estimate the mortality as less than 1 to 10,000 . . . " These figures are impressive.

The book is conservative, sane, and well written, and it thoroughly covers the subject. There is a bibliography, a list of authorities, and a subject index.

Surgeons, and especially anesthetists connected with hospitals, would do well to own the book and reread parts of it many times.

UROLOGY. By Daniel N. Eisendrath, M.D. and Harry C. Rolnick, M.D. Fourth Edition. Philadelphia, 1938. J. B. Lippincott Company. Price $10.00.

In a new format, and thoroughly revised and reset, this work comes to us in its fourth edition, attesting to its popularity and worth. The authors' first sentence, in their Preface, reads, "Our aim in this book has been to simplify the subject of urology for teaching purposes." And they have lived up to this aim to the letter.

It is not necessary to recite in detail the many changes made in this edition. Suffice it to say that this work is distinctly up to date, sane, and, while easily read, is scientific throughout. We would especially draw attention to the chapters on anomalies of the urinary organs and on seminal-vesiculitis.

This is a big book of 1,061 pages, beautifully made, with illustrations well above the average (750 black and white and twelve in color). The index is complete.

The authors and publishers deserve to be complimented for making a work of this type available to the profession.

HUMAN PATHOLOGY, A TEXTBOOK. By Howard T. Karsner, M.D. With an introduction by Simon Flexner, M.D. Fifth Edition, Revised. Philadelphia, 1938. J. B. Lippincott Company. Price $10.00.

A work that goes into a fifth edition just must be far above the average, since otherwise it would long since have languished and been forgotten. It is therefore more or less superfluous to tell our readers that Dr. Karsner has done an excellent piece of work in his "Human Pathology." Long, long ago this book's reputation was made (the first edition came out in 1926), and from time to time the author has revised his text, adding new material and altering various chapters.

In this edition, several new illustrations have replaced older ones and six new plates have been inserted to amplify the text. Dr. Simon Flexner has written an introduction, and the general text has undergone extensive revision.

The book is 1013 pages long, has 443 illustrations in black and white, and eighteen illustrations in color. References are given at the end of each chapter. There is an ample index.

This fifth edition of a recognized work leaves nothing to be desired.

ESSENTIALS OF PATHOLOGY. By Lawrence W. Smith, M.D. and Edwin S. Gault, M.D. With a foreword by James Ewing, M.D. New York, 1938. D. Appleton-Century Company. Price $9.00.

This work, designed specially for students, may well be studied by many physicians at large. The authors feel "that a thorough grounding in the fundamental principles of pathology is of greater value to the student than any attempt at encyclopedic presentation of the subject." With this basic thought in mind they have made this work *practical*, and have emphasized those things they believe to be fundamental and essential. In all controversial topics they have chosen the "middle course."

The pathology of the parasitic infestations is given more than usual space. Tumors, also, are emphasized to a great extent, as they should be. The various topics in the chapters follow a more or less uniform arrangement. In the beginning, there is a discussion of the subject. This is followed by selected illustrative case histories, complete with their associated gross and microscopic pathology.

As a further aid to the reader the pictorial method has been used. A number of roentgenograms and gross pathologic pictures have been included "in an attempt to correlate the appearance of the lesions with the microscopic histology."

This work of 886 pages, with 679 illustrations (many in color), a short foreword by the internationally known James Ewing has, in addition to the usual index, an index of the cases and associated illustrations. Also, blank pages for notes have been inserted.

A worthwhile, solid work. The publishers have offered the profession a large, typographically beautiful book at a very reasonable price.

DISEASES OF THE EAR, NOSE AND THROAT. By Francis L. Lederer, M.D. Philadelphia, 1938. F. A. Davis Company. Price $10.00.

This large book (835 pages) was written to meet the needs of students and general practitioners as well as to serve teachers and specialists. The author tells us that the teachings of Alexander, Beck, Hajek, Neumann, Pierce and others influenced the presentation of fundamentals of diseases of the upper air passages. The author also has drawn freely upon the world literature.

Certain terms, such as "Ménière's disease," "catarrh," "cardiospasm," "otosclerosis," and others have not been eliminated because they are still in usage. The author writes that a standardized nomenclature is not yet available.

The subject is covered to the minutest detail. Clearly written and well illustrated, the book contains nearly 500 half-tone and line engravings. In addition, there are sixteen full-page color plates. The author has purposely omitted a bibliography, but his index is ample.

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